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BOSTON STUDIES IN THE PHILOSOPHY OF SCIENCE
VOLUME XVI
Edited by Robert S. Cohen and Marx W. Wartofsky

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BOSTON STUDIES IN THE PHILOSOPHY OF SCIENCE

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VOLUME XVI

NORMAN GESCHWIND

SELECTED PAPERS ON LANGUAGE AND THE BRAIN

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PREFACE

Philosophers of science work not only with the methods of the sciences but with their contents as well. Substantive issues concerning the relation between mind and matter, between the material basis and the functions of cognition, have been central within the entire history of philosophy. We recall such philosophers as Aristotle, Descartes, the early Kant, Ernst Mach, and the early William James as directly inquiring of the organs and structures of thinking. Science and its philosophical self-criticism are especially and deeply united in the effort to understand the biological brain and human behavior, and so it requires no apology to include this collection of clinical studies among *Boston Studies in the Philosophy of Science*.

The work of Dr. Norman Geschwind, well represented in this selection, explores the relation between structure and function, between the anatomy of the brain and the 'higher' behavior of men and women. As a clinical neurologist, Geschwind was led to these studies particularly by his interest in those pathologies which have to do with human perception and language. His research into the anatomical substrates of specific disorders – and strikingly the aphasias – present a fascinating and provocative examination of fundamental questions which will concern not neurologists alone but also psychologists, physicians, linguists, speech pathologists, educators, anthropologists, historians of medicine, and philosophers, among others, namely all those interested in the characteristic modes of human activity, in speech, in perception, and in the learning process generally.

For philosophers and historians of science, questions about the characterization of human knowledge have come to be of major interest in recent years – its acquisition, its relation to perception and action, and their relation to the evolution of the brain. In contemporary developments of naturalistic and evolutionary epistemology, such philosophical issues are intimately linked with scientific investigations in linguistics, psychology and neurophysiology. We have had the good fortune to fol-

low Dr. Geschwind's studies over the past exciting decade of his research work, and he has on several occasions kindly participated in our colloquia, with that steady lucidity and rational directness which are among his fine qualities. Within these *Studies*, we have already published his critical and appreciative essay on 'The Work and Influence of Wernicke' which was introductory to our reprinting of Wernicke's classic and fundamental work of 1874 on aphasia, and also his influential survey 'Anatomy and the Higher Functions of the Brain' (both in *Boston Studies*, Vol. IV, 1969). It is most pleasant to bring the major papers of these years of Geschwind's work to a wider philosophical and scientific audience, and to anticipate the critical debates on conceptual and empirical questions which will follow.

Boston University Center for
Philosophy and History of Science,
April 1974

R. S. COHEN
M. W. WARTOFSKY

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INTRODUCTION

The papers in this volume, each one of which represents a publication of which I have been either a senior or junior author, cover a span of only 11 years. The papers chosen, moreover, reflect primarily a concern with one apparently limited approach to the problems of defining those features which most distinctively set off man in the animal kingdom. This restricted approach is one which concentrates on the anatomical organization of the brain, the evolutionary changes of which have determined the special biological adaptiveness of man.

This brief span of little over a decade has witnessed a remarkable change in attitudes concerning the area of brain-behavior relationships. My own background had led me into it through medicine, and in particular the field of clinical neurology. It is worth recalling what the state of the art was in 1961. Until the first World War the accomplishments in the field of understanding the relationships of the brain to language had been rightly regarded as among the brightest treasures of the discipline of clinical neurology, and the list of major contributors to it would have included a majority of the creators of the discipline – Broca, Meynert, Flechsig, Jackson, Wernicke, Liepmann, Lichtheim, Dejerine, to mention only a few.

Yet by the 1950's interest in this area had nearly vanished from the field of clinical neurology. There were a few exceptions, such as Luria in the Soviet Union, but much of his major work has been published only recently. In all of the French, German, and English-speaking worlds there were probably not a dozen neurologists for whom the higher functions of the brain represented a major interest. Indeed the word aphasia barely appeared in the neurological literature. Only 5 years ago the chairman of the program committee of a major neurological meeting complained that there were not enough papers to fill a half-day session on behavior. Furthermore, outside the field of clinical neurology there was little interest in the field of the relationship of anatomy to the higher functions or to their disorders.

As I have already said there has been a dramatic change within the last

10 years. Interest in aphasia and in disorders of the higher functions has grown rapidly from within and outside neurology. Papers in this area have increased sharply in numbers and quality. Not only have neurologists partaken of this revival of interest, but linguists, psychologists, speech pathologists, anthropologists and philosophers have come to be aware of the importance of the structure and evolution of the brain in understanding the biology of man.

The papers in this volume stress primarily the attempt to bring together anatomy and function. I do not wish to suggest that this is the only fruitful approach, and I have every reason to believe that new insights will come from linguistics and psychology. The past neglect of the insights to be obtained from anatomy is perhaps a major justification for the stress on this appraoch.

It is worth remembering that our understanding of the mechanisms of the disorders of the higher functions of the human brain is not merely a road to the clarification of major problems in biology, psychology, evolution, and philosophy. The acquired aphasias and the intellectual deteriorations of age remain major afflictions of mankind. The history of medicine has repeatedly taught us that advances in knowledge of structure and physiological mechanism have been followed by new and powerful methods of treatment. I believe that such advances will be well within our grasp in the next generation.

Norman Geschwind
James Jackson Putnam Professor of Neurology,
Harvard Medical School

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The author wishes to thank his many co-workers over the past years without whose invaluable help the papers contained in this volume would never have been published. In particular he wishes to express his gratitude to the co-authors of the following papers:

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'Language-Induced Epilepsy' – Dr. I. Sherwin
'Shrinking Retrograde Amnesia' – Dr. D. F. Benson
'Dichotic Listening after Section of Neocortical Commissures' – Mr. R. Sparkes
'Isolation of the Speech Area' – Dr. F. A. Quadfasel and Dr. J. M. Segarra
'Human Brain: Left-Right Asymmetries in Temporal Speech Region' – Dr. W. Levitsky.
'Developmental Gerstmann Syndrome' – Dr. D. F. Benson
'The Alexias' – Dr. D. F. Benson
'Writing Disturbances in Acute Confusional States' – Dr. F. Chédru
'Conduction Aphasia' – Drs. D. F. Benson, W. A. Sheremata, R. Bouchard, J. M. Segarra and D. M. Price
'Apraxia and Agraphia in a Left-Hander' – Drs. K. M. Heilman, J. M. Coyle, and E. F. Gonyea

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CHAPTER I

THE ANATOMY OF ACQUIRED DISORDERS OF READING

PREFACE

Like most neurologists of my generation I had been trained to an overwhelming skepticism toward the view that there were highly characteristic aphasic syndromes associated with different lesions of the brain. Even more forcefully I had accepted the view that any attempt at 'explaining' the syndromes on the basis of anatomy was a futile endeavor. Although Sir Charles Symonds had during my years at Queen Square stood almost alone against this view, I had been so caught up in the standard approach that I disregarded his perceptive teachings.

I had first heard about aphasia during Dr. Marcus Singer's outstanding neuroanatomy course at the Harvard Medical School. It was a common experience for students to have their interest aroused by the disorders of language, but despite this fact, nearly without exception they turned away from the field because there seemed to be no obvious way to become involved in research from the anatomical, physiological, or biochemical point of view. It is true that vigorous and productive research along predominantly psychological lines was being carried out by such workers as Benton, Goodglass, Hécaen, Milner, and Zangwill, but the conventional 'medical' approaches seemed to be futile.

By 1961 I found myself perplexed by the general rejection of anatomical approaches in contemporary writings contrasted with their support by so many of the great classical neurologists. I therefore decided to study the ideas of the classical 'localizationist' school by reading their own writings rather than by reading the interpretations of later hostile authors. It was my intention to decide for myself whether the repudiation of the classical views was indeed justified.

As it happened the very first paper that I read turned out to have a major impact on my thinking. It was Dejerine's paper describing the first post-mortem on a case of pure alexia without agraphia, of which my Chief at

the Boston Veterans Administration Hospital, Dr. Fred Quadfasel, had an original reprint.

The impact of the paper was multiple. In the first place the description was so lucid that it was immediately clear that this 'pure' syndrome must exist, despite the insistence by some modern writers that these selective syndromes 'could no longer be seen' (a statement implying that the earlier descriptions had been grossly in error). Indeed it was obvious that Dejerine's standard of examination was superior to that of most modern students of aphasia. Furthermore it was a shock, but a salutary one, to discover that even so masterful a paper had been neglected by later writers, or grossly misquoted. Henry Head (1926) the often cited critic of classical approaches, did not even list it in his bibliography. This paper was therefore instrumental in making me aware of the inaccuracy of most of the histories of aphasia in English.

The paper not only gave an anatomical interpretation of a behavioral syndrome, but it contained the first definite description of a callosal syndrome. I therefore realized that the common teaching in 1960 that callosal syndromes had not been observed in man was incorrect. I also realized that the standard accounts of localization had considered the doctrine of centers, but had generally totally neglected the classical stress on connections between cortical regions.

Within a few weeks of reading this paper I saw my first case of alexia without agraphia and only a few weeks after that the callosal case later published by Edith Kaplan and myself.

The following paper 'The Anatomy of Acquired Disorders of Reading' was given at a conference on dyslexia organized by Dr. John Money. It gave me my first opportunity to present my newly forming ideas on syndromes of disconnection, i.e., on syndromes produced by lesions of cortico-cortical connections. Needless to say, I was somewhat nervous, particularly since Oliver Zangwill, Professor of Psychology at Cambridge and one of the leading figures in the study of the higher functions was to discuss my paper. As it happened his comments were very generous. We had a long discussion of some of the applications of the idea of disconnection and out of this came the suggestion by Zangwill that I write up my ideas. Out of this arose 'Disconnection Syndromes in Animals and Man'.

The problem of pure alexia is, of course, a fascinating one in itself.

It has a special interest for me because of its importance in forming my approach to the higher functions.

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THE ANATOMY OF ACQUIRED DISORDERS OF READING*

1962

To most educators and pediatricians, the term *dyslexia* means the failure to acquire the ability to read; it is this disturbance which is the focus of interest of this volume. It is worth recalling that historically the first reading problems to have been investigated were cases of the *loss* of the ability to comprehend written language, and it is therefore common practice among neurologists to distinguish *acquired* dyslexia or alexia from their *congenital* counterparts.

The distinction between these acquired disturbances and the congenital dyslexias is not an academic one. Remarkably restricted lesions in the left cerebral hemisphere of the adult may cause permanent severe disabilities. By contrast, total destruction of the left hemisphere in early childhood ordinarily does not prevent acquisition of language. It seems clear that the distribution of pathology in the congenital disturbances of language must involve something other than what is involved in the aphasias of adults.

Very uncommonly, the acquired disabilities are of functional psychiatric origin (hysterical *blindness* is much more common). In nearly all cases the disability results from damage to the brain readily distinguished at post-mortem examination and the study of such lesions has been the most important technique for studying the parts of the nervous system involved in the comprehension of visual language in adults. An acquaintance with them is indispensable to any investigation of the apparently more elusive mechanisms involved in the failure to learn to read.

Three situations stand out prominently in which patients may lose the ability to read.[1] The most common is Wernicke's aphasia, in which the patient shows a severe incomprehension of both spoken and written language, together with a characteristic speech pattern (fluent speech which is often totally incomprehensible as the result of use of many distorted or incorrect words and extensive circumlocutions), and an inability to write correctly. A more restricted and less common syndrome is that of 'pure word blindness with agraphia'. The most restricted syn-

drome and the one whose mechanism I will discuss at some length is that of 'pure word blindness without agraphia'.

Before discussing this syndrome, the first question, oddly enough, must be whether I have a topic for discussion. Distinguished authors have disputed the existence of this entity. On the opposite side one finds equally distinguished adherents – Dejerine (1892); Bastian (1898); Holmes (1950); Symonds (1953); Warrington and Zangwill (1957); Hécaen, Ajuriaguerra, and David (1952); Alajouanine, Lhermitte, and Ribaucourt-Ducarne (1960). Some time ago those of us in the Aphasia Unit of the Boston Veterans Administration Hospital decided to review carefully individual case reports of this condition in the attempt to decide for ourselves whether it was justifiable to isolate the condition of 'pure word blindness without agraphia'.

We did not have to look far. The first reported case with a post-mortem examination is that of Dejerine (1892). Inexplicably this is a neglected paper. It is not mentioned in several works on aphasia. Where cited, it is often misquoted or even dismissed. Its value can be stated briefly. The careful description of the clinical picture has been confirmed more than once and it established an anatomical localization which has also been repeatedly confirmed. It is, in fact, a masterpiece of neurological clinico-pathological correlation.

Dejerine's patient was an extremely intelligent 68-year-old man who, on October 25, 1887, suddenly observed that he could no longer read a single word. He was examined first by Landolt (a distinguished ophthalmologist and a pioneer in physiological optics) and the original findings were confirmed by Dejerine repeatedly over the next four years.

The patient had a visual acuity of 8/10, spoke fluently without error, and understood all spoken speech. Objects were named perfectly, including pictures of technical instruments in a catalogue. He could identify his own morning newspaper by its form but could not read its name. On presentation he could not identify a single letter by name. The only written material he could read was his own name. Writing was correct, both spontaneously and to dictation, but what was written could not be read back. As Dejerine commented, his writing was rather like that of a blind man, larger than normal and with poor orientation of the lines.

Although the reading of isolated letters was impossible, the patient could identify them by name after tracing their contours with his finger;

if the examiner formed letters by moving the patient's hand through the air, he could name the letters produced in this way. This phenomenon had been described earlier in similar patients and many patients since then have shown the same finding.

A rather surprising contrast was the fact that even at the beginning of the illness, he could recognize individual Arabic numerals, but had trouble in reading several numbers simultaneously and in doing arithmetic calculations. With the passage of time all his difficulties in reading Arabic numerals and in doing even the most complex written calculations disappeared while his difficulties in reading letters persisted unchanged. Roman numerals were not tested. (Later observers have frequently confirmed the superiority in reading Arabic numerals in these patients, while Roman numerals seem to show the same difficulties as other letters.)

Like the other examples of this syndrome, this case showed a right hemianopia. Another frequently observed finding is a difficulty in color vision in the retained left visual field, but this is not mentioned by Dejerine.

The remarkable discrepancy between the loss of letters and preservation of numbers arouses one's interest in other visual symbols. The patient who had been a skilled musician now showed a total inability to comprehend musical notation, but could write a scale or particular notes to command. The ability to sing and play instruments was unimpaired.

The patient was observed carefully over the next four years. During this time he continued actively and very successfully in business, kept on writing, played cards skillfully, and learned and performed new music by ear. He had no difficulty in orientation even when going to strange parts of Paris.

On January 5, 1892, another cerebral vascular accident left him with paraphasic speech and incapable of writing. He died at 10 A.M. on the sixteenth of January and an autopsy was performed in the patient's home by Dejerine, twenty-four hours after his death.

Dejerine gave a careful description of the external appearance of the cerebral hemispheres and of the internal appearances as revealed by gross horizontal sections. There was no report of the microscopic findings in the lesions in this original publication, but these appeared in a later paper and in his monumental work on the anatomy of the brain. They fully confirmed the impressions he had drawn from observation of the gross specimen and supported his theoretical conclusions.

Two major lesions were found in the brain (Figures 1–4). There was the recent lesion (with which the patient had become paraphasic and agraphic) involving the angular gyrus and adjacent portions of parietal lobe and temporoparietal junction. It showed the characteristic features of a recent lesion: there were signs of both white and red infarction, in the absence of atrophy of the involved gyri, ingrowth of new vessels, yellowish coloration, thickening or adherence of the meninges. The older lesion (which is the one that is of major interest here since it is the one causative of the clinical picture I have been discussing) had caused destruction of the medial and inferior aspects of the occipital lobe. This showed the characteristic appearance of a lesion of considerable age with narrowing and atrophy of the gyri. The white matter of the occipital lobe was yellow and shrunken. In addition, there was a region of old destruction of white matter in the splenium of the corpus callosum.

In discussing the significance of the pathology, let us confine ourselves to the older lesion whose effects were apparent to Dejerine. He pointed out that for a word to be seen as language and not merely as an arbitrary design, there must be a connection between the visual centers in the occipital cortex and the language areas in the left hemisphere. But, in this patient, the destruction of the left occipital cortex and the resultant right hemianopia made it necessary for the patient to see only in his left visual field and hence, with his right occipital cortex. In this situation, for reading, it is necessary to have connections going from the right occipital cortex across the corpus callosum and to the left-sided language areas in the posterior temporal region. Dejerine argued that the extensive destruction of white matter in the left occipital lobe in this case would destroy the connecting fibers from the right occipital cortex. He thought that this destruction alone would suffice to disconnect the right visual area from the language areas without having to bring in the lesion of the splenium of the corpus callosum. There is good reason, however, to believe that the lesion of the corpus callosum did contribute significantly to the disconnection postulated by Dejerine. The anatomical evidence supports strongly the view that fibers from the occipital lobes to the corpus callosum run in the splenium where they are rather tightly packed.

It is worth commenting here that Dejerine's anatomical conclusions from this case are frequently misquoted. A frequent statement is that he attributed this syndrome to a lesion of the angular gyrus. In fact, he

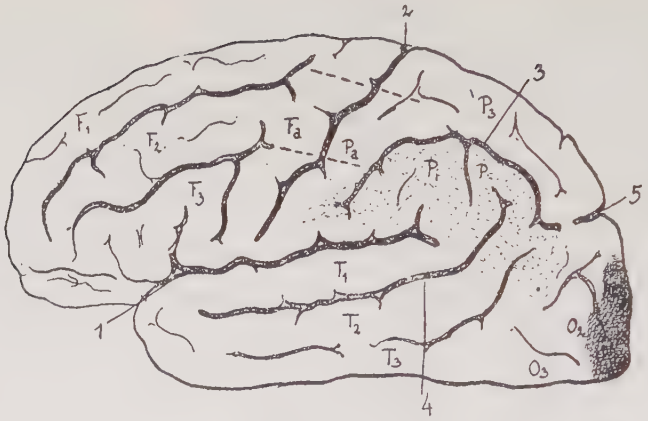

Fig. 1. Left hemisphere of Dejerine's patient, lateral view. The dark area in the occipital lobe represents the old region of destruction, the stippled area the recent lesion. 1 = Sylvian fissure; 2 = Rolandic fissure; F_3 = third frontal gyrus; T_1 = first temporal gyrus; Pc = angular gyrus. Reproduced from Dejerine (1892).

carefully denied this. He had shown a year earlier that a lesion of the angular gyrus caused the syndrome of pure word blindness with agraphia and indeed, he thought that it was specifically disconnection of the right visual cortex from the left angular gyrus which led to pure word blindness without agraphia.

There is one feature of the clinical picture which is very compatible with the idea of a specialized disconnection. This is the fact that although letters could not be read by this patient when presented visually, they could be 'read' when they were traced on his hand or when his hand was moved over the outline of a letter. This stimulation when presented to the right hand would go to the left sensory cortex and thence readily to the speech areas; when presented to the left hand, it would go across the corpus callosum (anteriorly to the splenium) from the right hemisphere and thus reach the speech areas. This preservation of tactile or kinesthetic 'reading' in pure word blindness without agraphia contrasts to its loss in pure word blindness with agraphia. The latter syndrome is the result of a lesion of a cortical area which somehow functions in the operations done on visual language; once destroyed, it is obvious that comprehension of written language is lost regardless of its mode of presentation; but, by a suitably placed lesion one can specifically disconnect from this cortical

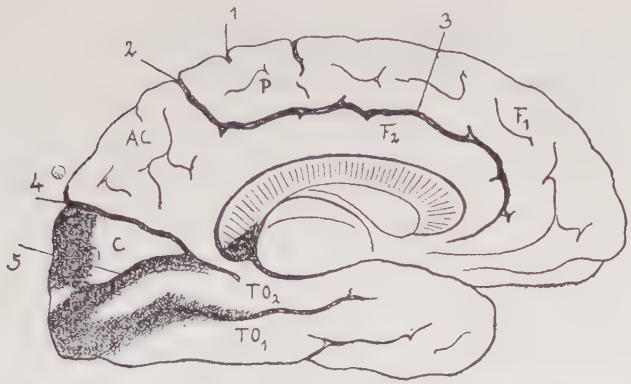

Fig. 2. Left hemisphere, medial view. The structure marked by radiating lines is the corpus callosum, the posterior end of which (the splenium) is seen to contain an area of old destruction. 5 = calcarine fissure, along the lips of which lies the visual cortex, which is shown as involved in the area of old destruction. Reproduced from Dejerine (1892).

area visually presented material alone, while preserving the transmission of somesthetically presented stimuli.

A few years later, Bastian (1898) reviewed two more cases (in addition to Dejerine's) which had come to post-mortem and had shown similar clinical and pathological findings and led him to defend essentially the same mechanism.

Foix and Hillemand (1925) advanced further evidence in support of a mechanism of disconnection and, in particular, of the importance of the lesion in the splenium. They had found that, in a case of destruction of the left occipital lobe with preservation of the splenium, there was no word blindness, despite the presence of the expected right hemianopia. By contrast, two cases which at post-mortem showed destruction of the splenium in addition to that of left visual cortex had manifested pure word blindness. The occurrence of this syndrome, dependent as it is on the concurrence of two lesions, is a result of the arrangement of the cerebral vessels. The posterior cerebral artery supplies not only the occipital cortex but the splenium of the corpus callosum. Hence, occlusions of this vessel are likely to lead to destruction of the two structures necessary for this syndrome. Had the posterior fifth of the corpus callosum been supplied like the anterior four fifths by the anterior cerebral artery, I suspect

that pure word blindness would be indeed an extremely rare syndrome.

The most recent confirmation both of the clinical picture and of pathology has been the work of Alajouanine, Lhermitte, and Ribaucourt–Ducarne (1960) who had six cases with the classical clinical syndrome, one of whom died showing the above distribution of pathology. They list several other workers who have also confirmed the pathological picture.

There is further evidence for the mechanism suggested by Dejerine and supported here. Cases of pure word blindness have been due most often to vascular disease and rarely to trauma. In the extensive studies of Teuber, Battersby, and Bender (1960) on 46 patients with war wounds of the occipital lobes, there was not a single case of word blindness. Furthermore, these authors point out that in World War I, no such cases were found by Marie and Chatelin or Wilbrand and Saenger. Professor Teuber has been kind enough to show me the notes on one patient seen after the completion of the above study who developed word blindness following trauma which, however, cleared in a few months.

The reason for the paucity of word blindness as a sequel of penetrating injury to the brain is, I believe, the fact that it would be extremely unusual to sustain injury simultaneously to the left visual cortex and the splenium of the corpus callosum. A missile penetrating the splenium would probably

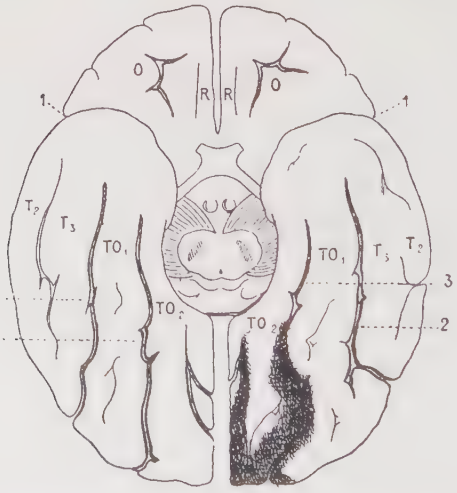

Fig. 3. Cerebral hemispheres, seen from below. Reproduced from Dejerine (1892).

Fig. 4. Diagrammatic horizontal cross section of cerebral hemispheres, illustrating the mechanism of pure word blindness without agraphia. NO = left optic nerve and NO′ = right optic nerve. C = left and right calcarine cortex, shown destroyed on the left (oblique lines); Pc = left angular gyrus (when destroyed as shown here alexia with agraphia results; this region is intact in pure alexia without agraphia); X = large lesion in white matter of left occipital lobe destroying fibers from the right visual cortex going to the left angular gyrus; cc = corpus callosum (although no lesion is shown here, a lesion here will be easily as effective as the lesion at X in disconnecting the right visual cortex from the left angular gyrus). Reproduced from Dejerine (1892).

enter the upper brain stem and almost certainly lead to the patient's death.

The evidence from neurosurgical procedures lends further support to the argument. Hécaen, Ajuriaguerra, and David (1952) describe seven patients who underwent occipital lobe resection, all very extensive. Every one of these patients developed pure word-blindness, but in every case the disability cleared up within six months. (It is interesting, however, that all of these patients found reading unpleasant after their recovery.) If pure

word blindness were the result of a lesion of some cortical region of the occipital lobe alone, permanent word blindness should have been seen in some of these cases. In contrast to the recovery of these patients is the fact that cases of this disorder that have a vascular origin have a very poor prognosis for recovery. The only differenee between the vascular and surgical cases is the preservation of the splenium in the latter. Presumably the fibers from the right occipital lobe to the speech areas are so tightly packed in the splenium that they are more effectively destroyed here than they are by even a very extensive lesion of the white matter of the left occipital lobe.

The above suggested mechanism has further implications. If the splenium alone were destroyed, words presented in the *right* visual field should be read normally, but the patient should be word-blind for words presented in the *left* visual field. Two papers have presented such cases. Thus, Trescher and Ford (1937) found that a patient whose splenium had been sectioned for removal of a third ventricle tumor could not read letters in the left visual field although the standard visual field examinations were normal, nor could she name wooden letters placed into her left hand. On the other hand, objects placed in the left hand were named correctly and two-point discrimination was normal in this hand. Maspes (1948) presented two further cases in whom the splenium of the corpus callosum was cut for removal of colloid cysts of the third ventricle. Both these cases developed alexia in the left visual field alone.

For the past fifteen years attribution of clinical symptomatology to lesions of the corpus callosum has been unpopular because of the negative reports of physiologists on the effects of callosal section and particularly as a result of the studies of Akelaitis and his co-workers (1941, 1943, 1944) on interruption of the callosum in man. However, Sperry (1961) and his colleagues have recently conclusively shown that dividing the callosum leads to profound changes in animals, if they are examined properly. Sperry showed that if one hemisphere is trained, the learned material is transferred to the opposite hemisphere by way of the corpus callosum. After section of the callosum, new material presented to one hemisphere is not transferred to the opposite side. In man, the testing situation is simpler than in the animal since language skills remain confined to the left hemisphere in normals. Hence, immediately following callosal destruction, appropriate testing should reveal defects if the right hemisphere is

tested with language tasks. The effect of posterior section is to cut off the right occipital lobe from the language areas. Sections further forward should have more profound effect on the limbs. Elsewhere we have described the effects of more anterior involvement of the corpus callosum (Geschwind and Kaplan, 1962). I would like especially to express my debt to Dr. Sperry whose work alerted me to the possible importance of callosal disconnection in humans.

The most serious objection to the mechanism discussed here arises from the aforementioned work of Akelaitis and his co-workers. In particular, he described six patients in whom the splenium was cut; in none of them was there any subsequent alexia in the left visual field. These six cases stand in contradiction to the case of Trescher and Ford (1937) and the two cases of Maspes (1948). There is at present no obvious means of explaining the discrepancy, although some suggestions may be made. One possibility is that the sections performed on Akelaitis's cases were less complete than those done on the patients of the other authors, but since none of the patients has had a post-mortem examination, this cannot be verified. Maspes pointed out that in five of the six cases studied by Akelaitis, cerebral lesions had been present from early childhood. Language skills might not have been sharply localized in the left hemisphere in these cases and callosal section would therefore have shown little effect. All of Akelaitis's cases were epileptics, while none of the contrasting group were. Could epilepsy favor the opening up of alternative pathways? This is a rather hazardous speculation which can only be mentioned here. Clearly, further observations are needed.

In this final section, I would like to turn to some more remote implications of the interpretation of pure word blindness without agraphia that has been defended here. First are its implications for the general problem of interpretation of the aphasias. Neurologists have frequently assumed that 'mixed' aphasias were the result of mixtures of 'pure' lesions. But, even the patient with pure word blindness with agraphia does not have the lesion of pure word blindness without agraphia 'mixed' with another lesion. Very pure lesions may result only from disconnection from the language areas and the possibility still remains that lesions directly in the speech areas can never produce a 'pure' syndrome (in the sense of an aphasic disorder confined to a single modality). This view gains added support from the fact that many researches over the years have

supported a similar mechanism for pure word deafness, i.e., a lesion of the left Heschl's gyrus, with destruction of callosal fibers from the right Heschl's gyrus, with no lesion in speech cortex. Hoff (1961) is the most recent investigator to have found pathological evidence for this mechanism.

The next point I would like to discuss is an apparent difficulty which arises from the mechanism for pure word blindness suggested above. Professor Davis Howes called my attention to the fact that none of the published cases of pure word blindness has had the symptom of so-called optic aphasia. By this term is meant the inability to *name* a seen object although the subject recognizes it (as proven, let us say, by his ability to select it afterwards from a group), with retained ability to name objects by feel, sound, smell, or taste. If indeed the right occipital lobe is disconnected from the language areas, how can the subject *name* seen objects? Let me sharpen this point by noting that in the patient mentioned earlier studied by Mrs. Kaplan and myself, who shows evidence of anterior callosal disconnection, one finds an inability to name *letters* placed in the left hand, i.e., a 'tactile alexia' of the left hand. He also shows an inability to name *objects* placed in the left hand although he can identify them nonverbally, i.e., he has the analogue to 'optic aphasia' which has been called 'tactile aphasia'. Why does 'optic aphasia' not accompany pure word blindness? One possible explanation is the straightforward anatomical one, i.e., that different pathways are used in naming an object, or more likely, that the sight of an object is much richer in associations than that of a letter and that thus eventually more anterior parts of the right hemisphere are stimulated whence pathways are found across the corpus callosum. Adolf Meyer (1950) supported essentially this latter version of the anatomical explanation.

There is another approach to the problem of the preservation of object-naming. Dr. Davis Howes and I have recently had the opportunity to study a patient with pure word blindness without agraphia who presented the classical clinical syndrome. In his paper in this symposium, Dr. Howes describes the studies that we have conducted, and in particular, he discusses the comparative data on object- and word-naming respectively. One possibility which emerges from these data is that while object- and word-naming may use the same pathways, object-naming may survive partial damage more effectively. The data do not clearly decide between the

anatomic and functional explanations and this remains an interesting area for further investigation.

In closing, we can turn to the question of the possible relevance of what has been presented here to the problem of 'congenital dyslexia'. I think one can say with some assurance that the lesion described here is not the cause of congenital dyslexia. Children with this disturbance do not have hemianopias. Furthermore, even if such a lesion did rarely occur in a child under the age of six, I doubt that it would impair his ability to learn to read, or if he were already reading, I believe that the disability would be transient. The reason for this is the tremendous ability of the youthful nervous system to find alternative means of compensating for damage to localized areas, a faculty much attenuated in the adult. However, I suspect that I could guess the type of explanation that Dejerine *might* have given for congenital dyslexia. He would have argued that the angular gyrus acts in some specific way to process visual language, and that *bilateral* maldevelopment of this region would be the minimum substrate for difficulty in learning to read. Is this the true explanation? I do not believe that an answer is available. I would only suggest that we keep in mind the lesson to be learned from Dejerine of the importance of interweaving our functional with our anatomical knowledge.

APPENDIX

This brief appendix is designed to aid the reader of this paper who lacks a technical background in neuroanatomy. The reader should refer to the illustrations given in the paper while reading this section.

The two halves of the brain (the *cerebral hemispheres*) are connected by a large bundle of nerve fibers called the *corpus callosum*, which can be seen on the inner surface of the divided brain. The posterior end of this structure is called the *splenium*. The cerebral hemispheres consist of an outer mantle of *gray matter* which is called the *cortex* which consists of *nerve cells* and a central core of *white matter* which consists of nerve fibers coming from or going to the cortex. The white matter carries to appropriate areas incoming sensations from the periphery, interconnects different parts of the cortex and carries impulses to muscles out of the cortex.

There are certain distinct specializations of the cortex. Visual impres-

sions go to the posterior end of the cerebral hemisphere, the *occipital lobes*, with the right side of the visual field being represented in the left occipital cortex and the left side in the right occipital cortex. Language functions are carried out only on the left side of the brain in most people in the areas marked in Figure 1 as F_3 which is in the *frontal lobe*, in the area marked T_1 which is in the *temporal lobe*, and in the area marked P_c (the *angular gyrus*) which is at the junction of the temporal, parietal, and occipital lobes.

The word *hemianopia* refers to a loss of vision in one half of the visual field. The *left* half of each retina receives the image from the *right* half of the visual field, since the incoming image from the visual field is inverted by the lens of each eye. The left half of each retina is connected to the left occipital lobe of the brain. Thus, a right hemianopia (for each eye) signifies impaired perception in the right visual field and results from destruction of the visual cortex in the left occipital lobe and vice versa for a left hemianopia.

NOTES

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In the ensuing discussion, a certain number of neuroanatomical terms will be used that may be unfamiliar to those without special training. Such readers are advised to begin by reading the Appendix to this paper, p. 15, where a brief outline of the necessary information is presented.

[1] Throughout this presentation the word *read* is used in the narrow sense of 'ability to comprehend language presented visually' and not at all in the sense of 'ability to read aloud'. Even in the neurological literature the two meanings are often confused. Many patients who cannot read aloud comprehend the written word correctly.

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CHAPTER II

RANDOM REPORTS: HUMAN SPLIT-BRAIN SYNDROMES

PREFACE

In early 1961 I had become aware, through reading Dejerine, (as discussed in the introduction to the preceding paper) of the occurrence of callosal syndromes in man. Within a few weeks I had seen my first case of alexia without agraphia. Shortly afterwards Edith Kaplan called another remarkable case to my attention. She had been examining a patient who had undergone a left frontal lobectomy for a glioblastoma and who had developed a striking grasp reflex in the right hand only, but not in the left. She decided that this would be an excellent case to test the assertions in an old paper of Bouman and Grünbaum that the grasp reflex caused typical defects in the mechanical characteristics of writing. She expected the patient's own uninvolved left hand to act as a control for the right hand which had the grasp reflex. To her astonishment she found that the patient wrote normally with the right hand and aphasically with the left hand. She told me about the case and since it struck me that this must be a callosal syndrome we began our joint investigation.

At this period the studies of Myers and Sperry had made the scientific world aware of callosal syndromes in animals, but there was still unawareness of their proven existence in man. The patient whom Edith Kaplan had discovered thus turned out to be the first modern human case of the syndrome of the anterior four-fifths of the corpus callosum, and also the first to come to post-mortem examination. We presented this case at the Boston Society of Neurology and Psychiatry on December 14, 1961. The brief report of this meeting in the New England Journal of Medicine is reprinted here because of its historical interest, as well as the subsequent fuller account in *Neurology*. As is well-known, Dr. Vogel later carried out the first of his callosal sections for epilepsy, and the studies by Sperry, Bogen, Gazzaniga and later collaborators confirmed again the presence of human callosal syndromes.

As I have noted ours was the first 'modern' case only in the sense that it

was the first such report after the awakening of interest in the syndromes of callosal disconnection in animals. We rapidly discovered that we were far from being the first to describe such patients. While Dejerine had pointed out the syndrome of the splenium in 1892, Liepmann had in the early 1900's described the syndromes of the anterior four-fifths of the corpus callosum. What was astonishing was the fact that this work had been so grossly neglected. It was published in widely-read journals and received wide acclaim in its day. Indeed it was discussed fully in Lange's (1936) article contained in the standard German neurological reference, the Bumke-Foerster Handbuch. Furthermore Liepmann's results had been reported with Otto Maas, who was still alive in the 1950's and had been confirmed by a whole array of authors such as Kurt Goldstein, who had himself described a callosal syndrome in 1908. Despite this fact, the students of Kurt Goldstein whom I met were generally unaware of this fact.

I was again made aware not merely of how inaccurate most of the histories of the higher functions were, but also that important confirmed scientific observations could almost be expunged from the knowledge of contemporary scientists. My presumption is that this must occur in other fields as well. The reasons for this phenomenon were fairly standard: neglect of work written in a foreign language, neglect of work done by someone in a different field, excessive reliance on the authority of certain towering individual figures.

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RANDOM REPORTS:
HUMAN SPLIT-BRAIN SYNDROMES*
1962

Until about 1940 callosal syndromes were widely accepted among neurologists. The rejection of such syndromes followed negative reports from physiologists on callosal section in animals and particularly the negative reports by Akelaitis on human callosal section. Recently, however, Sperry and his co-workers have proved that by means of new technics of examination, dramatic results can be shown to follow such sections. This work aroused the interest of the author in the possibility of human hemispheric deconnection syndromes. The paper presented evidence that pure word blindness without agraphia results from a lesion of the left visual cortex and splenium, relying on reported autopsied cases and on the extreme paucity of such cases after trauma or surgery. In the following paper a case that the authors thought could best be interpreted as hemispheric deconnection was presented. Objections were considered to the views presented in both papers, in particular the discrepancies between the authors' conclusions and those of Akelaitis.

In the second paper it was pointed out that cases of corpus-callosum lesions in human beings reported in the early 1900's were described as producing apraxia, apractic agraphia and astereognosis, all on the minor side. The authors reported the results of controlled experiments with a patient (suffering from a proved glioblastoma) who by conventional testing methods, utilizing verbal stimuli or requiring verbal responses, showed the clinical picture described by the earlier investigators. However, new testing methods revealed the inadequacy of employing such concepts as astereognosis, apraxia and agraphia as an explanation for the phenomena observed.

Their findings were that the patient responded correctly when stimulus and response were confined to the same hemisphere, and incorrectly when the stimulus was presented to one hemisphere and the response was required from the other. The authors believed that the patient behaved as if his cerebral hemispheres were disconnected and that a lesion of the corpus callosum was the most likely cause.

In the discussion Professor Patrick Wall asked about cases of agenesis of the corpus callosum. Dr. Geschwind replied that probably such patients would show none of the disturbances that the speakers had described because of the early age at onset and because speech might well not be lateralized. Professor Hans-Lukas Teuber commented that he personally had seen the patient and wished to congratulate the speakers on their analysis of the case. He raised the possibility that not disconnection but perhaps interference between the hemispheres was present. Dr. Geschwind pointed out that there was no positive evidence for interference in their case since as long as stimulation and response were confined to a single hemisphere the subject performed correctly.

Dr. Fred A. Quadfasel congratulated Mrs. Kaplan on her initial examination in which she had uncovered the aphasic difficulty in writing with the left hand alone that had led to further investigation of this case.

NOTE

* Based on two papers, the first, 'Anatomy of Pure Word Blindness without Agraphia', presented by Dr. Norman Geschwind, and the second, 'Human Anterior Deconnection Syndrome', by Mrs. Edith Kaplan and Dr. Geschwind at the meeting of the Boston Society of Psychiatry and Neurology held on December 14, 1961, with the ensuing discussion.

This paper originally appeared in the following form, without an author's name:
'Random Reports: Human Split-Brain Syndromes', *New England Journal of Medicine* **266** (1962) 1013.

CHAPTER III

A HUMAN CEREBRAL DECONNECTION SYNDROME*

A preliminary report

1962

ABSTRACT. A case of a 42-year-old man with a glioblastoma multiforme, who postoperatively was found to present many unusual findings, is reported. The patient's routine neurological examination showed a right hemiplegia worse in the leg with a very marked grasp reflex in the right hand and mild position sense loss on the right side. There were definite signs of intellectual deterioration.

Writing with the left hand was incorrect, both spontaneously and to dictation, while writing with the right hand was normal except for some disturbances caused by the grasp reflex. He named objects placed in the left hand (concealed from vision) incorrectly; he could select them afterward with his left hand by touch or pointing; and he could draw the object afterward with his left hand. Even while giving an incorrect verbal description, he could demonstrate correctly the use of the object being held in the left hand. If an object was placed in one hand (concealed from vision), he could not select it from a group or draw it with the other hand. He frequently performed verbal commands incorrectly with his left hand.

The authors feel that the simplest explanation of the phenomena is that the patient behaved as if his 2 cerebral hemispheres were disconnected and that the probable cause of this was a lesion of the corpus callosum. Alternative hypotheses and objections are considered, and a brief review is given of relevant earlier clinical observations as well as of the recent work of Sperry on callosal deconnection in animals.

We propose in this paper to present a patient whose clinical picture appears to us to be most simply explainable by a partial deconnection of the two cerebral hemispheres. He appears to behave as if there were 2 nearly isolated half-brains, functioning almost independently.

In the early years if this century several cases were described which showed some of the phenomena that are present in our patient. Sittig (1931) reviews these cases in his monograph on apraxia. The earlier workers generally described these cases as showing apraxia and apractic agraphia of the left side and left-sided astereognosis and attributed these findings to lesions of the corpus callosum.

In the 1940's considerable doubt was cast on the role of the corpus callosum by the extensive studies of Akelaitis (1944) and his co-workers on humans whose corpora callosa had been sectioned surgically (to prevent the interhemispheric spread of seizures) and who subsequently presented virtually no abnormalities. Earlier physiological work in animals also

failed to show convincing disturbances. Bremer, Brihaye and André-Balisaux (1956) have reviewed this literature. By contrast, in the last five years, the work of Sperry and his co-workers has convincingly proved that in animals, section of the callosum produces behavior which is most simply explained as resulting from deconnection of the 2 hemispheres (Sperry, 1961). It was Sperry's work which alerted us to the possibility of deconnection syndromes in man.

Our patient shows behavior similar to that described by the earlier workers. In addition we have observed several manifestations not previously mentioned. Detailed anatomical confirmation of the localization of the lesion is not yet available. The patient continues to be studied actively at this time. However, in view of the unusual character of these findings, we are presenting this brief clinical description as a preliminary report in the hope of stimulating other workers to look for similar cases and to investigate their anatomical substratum.

I. CASE REPORT

P. J. K. (BVAH U-53490), a 41-year-old white, married police officer, was admitted to the Boston V.A. Hospital, Neurology Section, on March 2, 1961. One month before admission the patient had begun to develop dull headaches, primarily over the left orbit, lasting several hours, recurring 3 to 4 times a week, and frequently associated with nausea and vomiting. The members of the family had over the previous few months noticed increasing behavioral changes manifested by indifference; apathy; forgetfulness; diminished alertness; confusion for dates, events, and people; diminution in personal neatness; and increased friction in interpersonal relations, particularly at work.

The patient had no significant history of birth or childhood illness. He had been graduated from high school at 18, had served in the Navy from 1941 to 1945, and since 1949 had been a policeman. Past history and family history were otherwise not relevant.

General physical examination revealed a well-developed, well-nourished man. The temperature was 98.6°F., blood pressure was 110/90, and pulse, 80. There were no significant abnormalities in the remainder of the general examination.

Neurological examination revealed a patient who was alert, cooperative,

and oriented in all spheres. He exhibited inappropriate jocularity against a background of general apathy. He had no insight into his illness and appeared unconcerned about it. He kept repeating questions as if he did not quite understand them. He could remember only 1 of 3 items after five minutes. He repeated 6 digits forward and 4 backward. He made many errors in subtracting sevens serially from 100. His proverb interpretations were very poor, being little more than restatements without interpretation. He did simple written arithmetic correctly but failed on more complex material such as multiplying 214 by 35. When asked to draw a clock face at a certain hour, he frequently reversed the large and small hands.

The patient was right-handed. Except for a few paraphasias and some hesitancy in naming objects, speech was normal. He drew a rough map of the United States on which his localization of many cities and states was correct except for New Mexico which was included twice in different parts of the Southwest. There was no right-left disturbance.

On examination of the cranial nerves, the optic disks were indistinct but without papilledema. Visual fields to confrontation showed a questionable binasal lower quadrant defect, but this was not confirmed by perimetry. The right pupil was slightly larger than the left, but both showed normal responses to light and convergence. Optokinetic reflexes were present bilaterally. There was questionable flattening of the left nasolabial fold. Cranial nerves were otherwise intact.

Motor examination showed normal bulk, tone, strength, and coordination throughout. Deep tendon reflexes were 1–2+ throughout but equal on both sides. Abdominals were 2+ bilaterally. Plantars were flexor, and grasping, sucking, and snout reflexes were said to be absent. Sensory examination showed no abnormality of touch, pin-prick, position sense, vibration, two-point discrimination or stereognosis. Stance and gait were both normal.

Laboratory examination showed a hematocrit of 45 per cent and a hemoglobin of 14.3 g. White blood count was 6200 with a normal differential. Corrected sedimentation rate was 15 mm. per hour; fasting blood sugar, 94 mg; blood urea nitrogen, 23 mg; prothrombin time, 100 per cent; bleeding time, 1 minute; and coagulation time, 18 minutes. Spinal fluid showed an opening pressure of 160 mm. and a closing pressure of 80 mm. The fluid was clear and colorless with no cells. Protein was 60 mg; sugar, 110; chloride, 118 mEq; colloidal gold, 011232100; and

zinc sulfate test, 4+. Serology was negative in spinal fluid and blood.

The electrocardiogram was normal. Visual fields by perimetry were normal. X-rays of the chest and skull were felt to show no definite abnormality. A pneumoencephalogram with 20 ml of air was consistent with a left frontal space-occupying mass. A left carotid arteriogram was consistent with a left frontal mass, probably a glioma.

Psychological testing on March 8, 1961, revealed a Wechsler Adult Inteligence Scale full scale I.Q. of 81 with a verbal I.Q. of 79 and a performance I.Q. of 87. On the Wechsler Memory Scale, he achieved a score of 87. Memory for visual forms showed impairment but copying was normal. The results of psychological testing were regarded as clearly consistent with intellectual deterioration.

On March 16, 1961, the patient underwent a left frontal craniotomy. On inspection, all of the visible left frontal lobe was markedly softened. A cyst deep in this lobe was aspirated through the middle frontal gyrus. A frontal amputation was performed revealing tumor in the depths. Because of edema, the anterior border of the corpus callosum was not identified. A large branch of the anterior cerebral artery on the medial surface of the frontal lobe was amputated. This was thought to be most likely the frontal polar artery. The ventricular system was not entered. The patient received 3 liters of blood and tolerated the procedure well.

The pathologist described the specimen as left frontal lobe, weighing 107 g, and identified the tumor microscopically as a glioblastoma multiforme.

Postoperatively the patient showed a dense right hemiplegia and a marked aphasia. He improved steadily and was returned to the Neurology Service. On the 49th postoperative day, neurological examination revealed a patient disoriented in time, but correctly oriented to place and person. He was apathetic but smiled most of the time. He confabulated in response to questions about the reasons for his admission to the hospital. There was impairment of recent memory, calculation, and abstraction. His speech showed at most a few mild paraphasic errors. He followed complex commands poorly. There was a dense weakness of the right leg and moderate weakness of the hand and arm with equal involvement of flexors and extensors. The finger-nose test was well done.

There was a mild tremor in the outstreched fingers of the right hand as well as a marked grasp reflex. The tendency to grasp was most marked

when the zone between thumb and forefinger was stroked. Traction on the arm further intensified the grasp. When the examiner's hand was brought near the palmar surface of the patient's right hand without actual contact, the patient's hand followed the examiner's.

In the left limbs, tone, power, and coordination were completely normal and there was no trace of grasping in either the left hand or foot. Deep tendon reflexes were symmetrically hyperactive and both plantars were flexor.

Sensory examination on the right showed a mild loss of position sense in the fingers and a denser loss in the foot. Vibration and stereognosis were intact and localization of touch was grossly accurate. Two-point discrimination was mildly impaired and pin-prick appeared to be mildly diminished. On the left side sensory examination was at first very confusing because the patient gave extremely variable reports. However, when for reasons which will become apparent later, technics for testing sensation on the left side were developed in which the patient was required to respond nonverbally (for example, pointing up and down in position sense testing, tapping once or twice for two-point discrimination), it could be shown that position sense, vibration, two-point discrimination, pin-prick, and touch localization were all normal in the left arm and leg. Discussion of stereognosis in the left hand will be deferred to the section on Results of Special Studies.

On May 22, 1961, it was discovered by one of us (E.K.) that he could not write with his left hand. At this time a program of special investigations was instituted, the results of which are given later.

Psychological testing on May 28 and September 13 showed no essential differences from preoperative testing.

The patient remained in the hospital until September 4, 1961, by which time he had improved considerably. He was oriented in all spheres but still had little insight into his illnes and showed unchanged intellectual impairment. The right hemiplegia had improved markedly. In the right arm there was only mild weakness of abduction of the right shoulder; in the right leg there was only mild weakness in flexion and extension at the knee and inability fully to dorsiflex the ankle. The patient could now walk unaided. The grasping responses were markedly diminished in all respects except that with the diminution of the paralysis of the right leg, a mild grasp reflex had now become apparent in the right foot. Improve-

ment observed during follow-up after discharge from the hospital will be discussed below.

II. RESULTS OF SPECIAL STUDIES

When it was discovered that the patient was unable to write correctly with his left hand, a program of carefully planned testing was set up. The results of these examinations are now given.

At the time of testing the patient showed no evidence of aphasia in his spontaneous speech although there was a mildly halting quality to his language productions. The patient showed no evidence of word-finding difficulty. There was at most a mild difficulty in comprehension of complex spoken or written material which was felt to be consistent with his intellectual impairment. He correctly repeated numbers, letters, words, and sentences presented verbally.

Writing with the Right Hand

When asked to write with this hand the patient produced normal sentences and made no errors in spelling. There were certain characteristic disturbances in the motor performance (Figure 1). His writing tended to get smaller with letters closer together; eventually one letter would be written over the preceding one (Figure 1b). In writing single letters or numbers he tended to keep going over the outline (Figure 1e). He would add extra loops when writing 3's or m's or three-looped figures (Figure 1d). Review of his preoperative writing revealed that similar defects had been present although they had passed unnoticed (Figure 1f, g, h). Bouman and Grünbaum (1930) in their study of patients with grasp reflexes described similar graphic disturbances.

Similarly, letters, numbers, words, and sentences were correctly written to dication. He copied all forms of written material correctly. When presented with printed material, he would copy it in script, which is the typical response of normal people to this task.

In typing with the right hand (using one finger), he produced his name and words to dictation with no errors (Figure 1c).

Writing with the Left Hand

By contrast, when asked to write with this hand he made many errors.

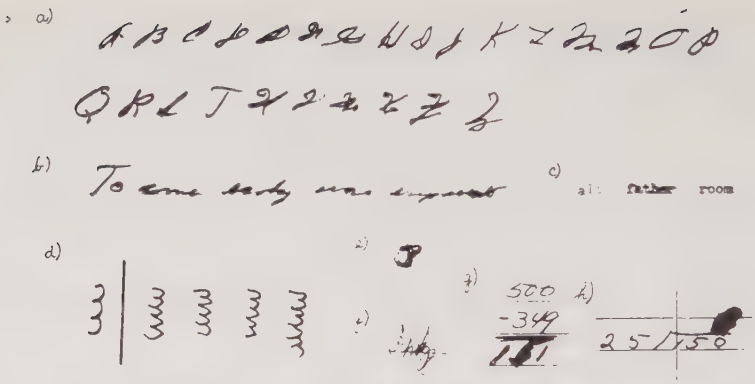

Fig. 1. Samples of performances with the right hand, demonstrating the effect of the grasp reflex: (a) the alphabet; (b) the sentence, "To come early was impossible" written to dictation: (c) the words, 'all', 'father', and 'room' typed with the right index finger; (d) to the left of the vertical line examiner's model three-looped figure, to the right of the line patient's successive attempts to copy the model; (e) the number '3' written to dictation; (f) the patient's preoperative writing of his first name; (g) and (h) the heavily written numbers are the patient's solutions to the problems written by the examiner (preoperative).

In writing the alphabet, he produced many incorrect or completely unrecognizable letters (Figure 2a). On dictation he would generally write a clearly recognizable letter or number but usually not the one demanded. When asked to write words and sentences to dictation, he would usually produce an unrecognizable scrawl (Figure 2b). On occasion, however, the response consisted of well-written but clearly incorrect words such as yonti for yesterday (see also Figure 2e, f). He copied printed and written material correctly but would reproduce printed material with block letters and not script. There was no trace of the motor difficulties seen in the productions of the right hand (Figure 2c).

In typing with his left hand, he could not produce his own name correctly nor could he type words correctly to dictation (Figure 2d).

When he had seen the incorrect productions of his left hand, he was quite astonished. However, spontaneous attempts to correct generally resulted in repeated difficulty (Figure 2d).

Object and Letter Identification with the Right Hand

With his eyes closed, the patient correctly and rapidly named objects and

cardboard letters placed in his right hand. Similarly, he could, after the object was taken away, correctly draw the object which had been in his hand. If letters or numbers were traced on his right hand, he generally named them correctly.

Object and Letter Identification with the Left Hand

By contrast, when objects, numbers, or letters were placed in the left hand, the patient (with eyes closed) generally named them incorrectly. The errors usually bore no resemblance to the stimulus object. For example, a ring was identified as 'an eraser', a watch as 'a balloon', a padlock as 'a book of matches', and a nail as 'an elastic'. The errors were not constant. Thus, on another occasion he called a ring 'a package of some sort'; a screw driver on one test was 'a spoon' and on another 'a piece of paper'.

It was striking to observe that when handling the objects he would move them actively about within his fingers and would focus on their salient features. For example, he inserted his finger into a thimble, ran his thumb

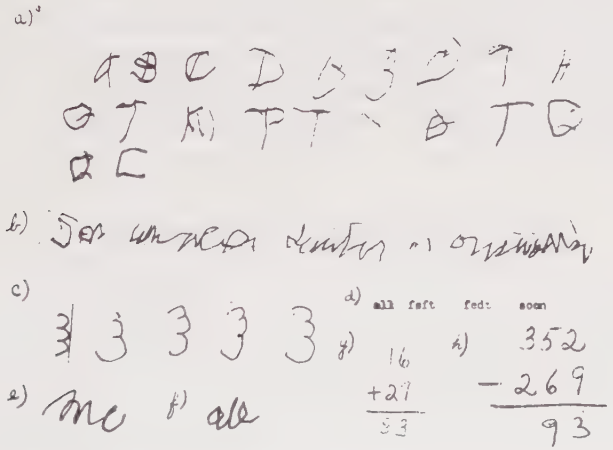

Fig. 2. Samples of performances with the left hand, demonstrating errors in language and calculation along with the absence of disturbances due to grasp reflex: (a) the alphabet; (b) the sentence, "To come early was impossible" written to dictation; (c) to the left of the vertical line examiner's model three-looped figure, to the right of the line patient's successive copies of the model; (d) attempts with left index finger to type the words, 'all', 'father', 'father' (the second being a spontaneous attempt to correct the first error), and 'room'; (e) attempt to write 'run' to dictation; (f) attempt to write 'go' to dictation; (g) and (h) the patient's solutions to the problems set by the examiner.

over the teeth of a comb, rubbed the bristles of a tooth brush, and retracted the point of a ballpoint pen. Despite his appropriate handling of the dominant features of these objects, he misnamed them all.

When it was made clear to him that he was to show how the object placed in his hand was to be used, the subject (with eyes closed) proceeded with little hesitation to manipulate the item correctly, usually giving simultaneously an incorrect verbal account. Thus, given a hammer, he made hammering movements correctly while saying "I would use this to comb my hair with it." Given a key, he went through the motions of inserting it into a lock and turning it but said that he was "erasing a blackboard with a chalk eraser." Holding a pair of scissors correctly, he made cutting movements in the air but said that "I'd use that to light a cigarette with." When his eyes were open he correctly identified the objects in his left hand.

Still another method was employed to study identification with the left hand. The patient, with eyes closed, was given an object in this hand.The object having been taken away, the left hand of the patient (whose eyes still were closed) was placed in a box containing a number of items including the test object. He selected the correct object. Similarly, with eyes open, he would without hesitation point to the object in a group without touching it. He was also able to make a correct drawing with his left hand of an object that had been previously placed in this hand and then removed.

Objects Placed in One Hand with Identification by the Other Hand. If an object or letter was placed in the left hand behind the patient's back, he generally was unable with the right hand (1) to select it correctly from a group of objects presented to him either visually or tactually, (2) to write the name of the object, or (3) to draw a picture of the object. For example, a letter which would have been drawn correctly with the right hand after having been held in the right hand was drawn incorrectly with the right hand if the letter was placed in the left hand. Similarly, if objects were placed in the right hand, the patient was usually incapable of performing any of these maneuvers with his left hand.

These results contrast with data given earlier in which we demonstrated that if an object was placed in one hand, it could afterward be identified by drawing or selection by the same hand. In addition, we may note here

that if letters were traced on the left hand, the patient could afterward trace them correctly on the ground with his left foot. If letters were traced on the right hand, he could not reproduce them correctly with the left foot. The right foot could not be tested in this way because of paralysis.

Ability to Perform Commands

When the patient was asked to perform actions with his right hand, he did so correctly and without hesitation. For example, he could draw a square, wave goodbye, salute, and pretend to brush his teeth or comb his hair.

When asked to perform actions with his left hand, he made many errors and even the correct responses were performed with a considerable delay. Thus, when asked to draw a square, he drew a circle. When asked to draw a triangle (which he had done earlier with his right hand without hesitation), he paused, asked if the examiner meant the figure with 3 corners, and finally drew the figure. When asked to point to the examiner with his left index finger, he pointed to his own eye with his left index and middle fingers. Immediately afterward he performed this action correctly with his right hand. Requested again to perform with his left hand, he pointed to the floor with his index finger. When asked to show how he would brush his teeth with his left hand, he on one occasion made the motions of lathering his face and on another went through the motions of combing his hair.

When asked to imitate the examiner's movements or to manipulate objects, the patient performed correctly with *either* hand.

Transfer of Learning

We attempted to perform a version of Sperry's experiment on transfer of learning (Sperry, 1961). We intended to have the patient learn (when blindfolded) a simple maze with the index finger of one hand, and then test the index finger of the other hand. In our normal controls such learning always transferred in 1 or 2 trials. However, this experiment could not be carried through successfully. The patient learned the maze with his left hand but was totally incapable of running the maze with the right hand. This failure to perform with the right hand appeared to be due to the grasping responses. The patient would either hold his finger fixed at one point or, in an effort to free it, suddenly jerk his finger erratically out of the maze.

Bilateral Movements

The patient could make circles in the air with both hands, tap with both hands, or perform alternating movements with both arms. It was observed, however, that in these symmetrically performed activities the two hands would on most occasions move at first at the same speed but after a time become desynchronized; on other occasions they moved at different speeds right from the onset.

Previously learned movements requiring the cooperation of both hands such as threading a needle, catching a ball, tying shoelaces, and dressing himself were performed without difficulty.

Parietal Lobe Testing

On the right side we have already noted evidence of mild position sense and two-point discrimination loss. On the left side both of these functions were normal. Pin-prick was identified correctly on both sides, but there was more withdrawal on the left. Touch and vibration were normal on both sides. Stereognosis, when appropriately tested for, was fully normal on both sides. Localization of touch was normal on both sides. Bilateral simultaneous stimulation gave highly variable results, but there was no consistent evidence of extinction on either side.

Using his right hand, he could correctly point out fingers on both hands and other body parts on both sides. With his left hand he made many errors on this test but no more than in performing any other movements to verbal command with this hand. He could correctly name parts of the examiner's body. With his right hand he made only 1 error out of 8 in a screening test of right-left orientation (indicating body-parts on the examiner and on himself). With his left hand, on the same test, he made 6 errors but again his difficulty was no more profound than in performing other verbal commands with this hand.

He showed no evidence of topographic disorientation. On one occasion he was being taken from his home to the hospital in the car of one of the authors. He suggested a complicated short-cut and correctly gave all the directions for following it. He had no difficulty finding his way around the hospital. On a map he correctly located Europe, the Atlantic Ocean, the Mississippi River, the Rocky Mountains, New York, Chicago, San Francisco, and Miami.

In setting a clock face he often reversed the large and small hands but

made no other errors. He copied complex stick figures perfectly with the model present. On one occasion he pointed out correctly to the examiner that he had been given one stick too few to complete the design. He made 50 per cent errors with the model absent. He performed normally on the Block-Design subtest of the Wechsler Adult Intelligence Scale. He drew and copied figures with either hand although on the right the proportions were poorer and he tended to make extra strokes. There was no evidence of neglect of either half of space in his drawings.

The patient dressed himself correctly and without difficulty. There was no neglect of the left side of the body. If neglect existed at all, it was manifested only on the right. He complained that his right hand was dead or useless and would use his left hand preferentially in many tasks (for example, in picking up objects from the floor). Objects placed in the left hand were always handled vigorously and correctly. The left arm showed no evidence of catatonic postures nor any responses of avoidance.

Simple written calculations were correct with the right hand, but he failed on more complex problems. With the left hand his calculations were grossly incorrect, even on the simplest problems (Figure 2g, h). His calculation difficulty with the right hand ran parallel to his poor performance on tests of abstraction such as proverb interpretation and similarities.

Course Since Discharge

The patient has been observed frequently in follow-up. At the time of this writing (two months after discharge), there have been some changes in the clinical picture. There have been some signs of progression in that the position sense loss in the right fingers is clearly worse, and he shows difficulties in identifying block letters placed in this hand. He makes many more errors in right-left testing. On a more complex test of finger identification than was used in the earlier testing, he still performs correctly with the right hand. He made 2 errors out of 10 on the most difficult subtest. This test is one in which the patient was asked to show on his body a pair of fingers simultaneously pointed to by the examiner on a model hand.

By contrast, his writing with the left hand has improved in that he produces fewer illegible words, but the words produced are still usually grossly incorrect. He makes fewer errors now in following verbal commands with the left hand.

III. DISCUSSION

In the early 1900's several cases sharing certain similarities with our patient were reported. In general, these case were described as showing an apraxia of the left side, agraphia of the left arm alone, and astereognosis on the left. Along with these findings, there were generally associated a hemiplegia, worse in the leg, and a powerful grasp reflex on the same side as the predominantly crural hemiplegia. We have critically examined these reports, and in a later paper we will present our analysis of them in detail. We would stress here that in our opinion some of these cases, when viewed in isolation, would be difficult to interpret. Thus, in Liepmann and Maas's (1907) famous case, Ochs, there was a dense hemiplegia involving the right arm as well as the leg. Since no testing for praxis or writing could be done except very rudimentarily on the right, one might have doubts as to the unilaterality of the apraxia and agraphia. Our view as to the possible difficulty of drawing conclusions from such a case coincides with that of Hécaen and Gimeno-Alava (1960). In Goldstein's (1908) case other difficulties arise. Here the right side was perfectly normal and apraxia, agraphia, and astereognosis were reported on the left. However, this patient had a left hemiplegia, worse in the leg, and a remarkably intense grasp reflex of the left hand. We agree with Denny-Brown (1958) that careful study is needed to insure that apparent dyspraxic difficulties are not due to a grasp reflex.

Despite these difficulties, there are cases such as those of Van Vleuten (1907) and Maas (1907) in which the hemiplegia involved the right leg and not the right arm; where, despite the grasp reflex in the right hand, there were no right-sided agraphic or apraxic difficulties; and where the otherwise normal left side was reported to show agraphia, and astereognosis.

The feature common to these early reports was a lesion in the distribution of the anterior cerebral artery, in some cases vascular, in some tumor, and in particular, in all instances involving corpus callosum. In many of these cases the localization was confirmed at postmortem examination; in the others the pattern of crural monoplegia with a marked grasp reflex in the homolateral hand (such as had generally been present in the autopsied cases) led to a similar clinical localization.

The early workers interpreted their data as being due to callosal decon-

nection. They believed that the left hemisphere was the leading hemisphere for movement and that the right hemisphere could not function properly in isolation. In the same manner, they felt that the left hemisphere was dominant for stereognosis and that the astereognosis of the left hand could be interpreted as due to separation of the right somesthetic cortex from the left.

Cases of this type have been described much more infrequently in recent years. Other cases of left-sided apraxia have been noted in which the associated clinical symptomatology has been quite different and strongly suggestive of right parietal disease (Hécaen and Gimeno-Alava, 1960).

Our own case shows clear clinical affinites with the early group in its association of what superficially appear to be purely left-sided difficulties together with right crural monoplegia and a right grasp reflex. Had we not introduced certain new methods of study, we might have interpreted the findings as those of left-sided apraxia, apractic agraphia, and astereognosis.

Our data clearly rule out asterognosis in the left hand. As long as verbal responses were avoided, identification was perfect. The patient was thus simply unable to name objects or letters placed in the left hand. It is important to stress that unless a patient is tested in this way, one can neither ascribe the difficulty to a right parietal lesion nor use the term 'astereognosis'.

Our patient's agraphia of the left hand cannot be regarded as being secondary to elementary movement difficulties. The patient drew very well with this hand, and indeed his drawing was better with this hand than with the right. The written productions often consisted of perfectly formed letters or numbers which were incorrect. His difficulties in forming words appeared equally in writing, using anagram letters, or typing.

In addition, our testing showed certain unusual features in the apparently apraxic manifestations. Thus, the poor performance of commands by the left side would appear to fit the classical picture. However, we could apparently induce an apraxic difficulty of the right side by placing objects (concealed from vision) in the left hand and asking him to draw or otherwise identify them with the right hand. This was parallel to the inability to identify with the left hand, either by touch or pointing

or drawing, objects that had been held (concealed from vision) in the right hand.

Certain interpretations of the data can be excluded. The possibility of hysteria or malingering must always be considered in cases with unusual manifestations, but this seems highly unlikely here. Classical hysterical manifestations were totally absent; the pattern of ability and disability was too complex, especially considering the patient's limited intellectual abilities; many features showed an affinity to the above-mentioned classical clinical pictures which could hardly have been known to the patient; and prolonged careful observation of his behavior militated against this interpretation. Similarly, the patient's intellectual impairment could not explain the differences on the 2 sides of the body. We will later discuss a possible indirect role of intellectual deterioration in the clinical picture.

The data do not support a parietal lobe syndrome either right- or left-sided as the cause of the difficulties. He showed some mild position sense and two-point discrimination loss on the right but no astereognosis on either side. There was no loss of two-point discrimination or position sense on the left. Dressing and topographic difficulties were absent; copying difficulties were not present; and there was no finger agnosia or other autotopagnosia. In particular, we were very careful to verify the absence of neglect of the left arm (if anything, there was a preference for this arm) or of the left half of space, and there was no evidence of avoiding responses or catatonic postures on the left. The course since discharge also supports this view. There has been evidence of progression of left parietal lobe signs: that is, increased position sense loss in the right hand, the appearance for the first time in this hand of an inability to identify block letters, and increased difficulties in right-left orientation. Yet this has been accompanied by an amelioration of many of the distinctive features of this case: improved writing and increased ability to follow verbal commands with the left hand.

Could the disturbances of the left arm be due to unsuspected involvement of the right hemisphere? We have already excluded the possibility of right parietal disturbance. Against the notion that there is involvement of motor regions of the right cerebrum is the total absence of left-sided weakness, grasping, or incoordination. On the right side, where such signs were present, the left-sided deficits were conspicuously absent. Even

if there were some heretofore unknown higher order reflex disturbance (similar to grasping or avoiding) in the left arm, it could hardly explain the aphasic nature of his writing difficulties and his inability to name objects placed in the left hand; nor could it explain the failures of the right hand to reproduce or identify material placed in the left hand.

It appears to us that the simplest description of this patient's most striking disturbances is that he behaved as if his two cerebral hemispheres were functioning nearly autonomously. Thus, we found that so long as we confined stimulation and response within the same hemisphere, the patient showed correct performance. If an object was placed in one hand when the patient had his eyes closed, the patient could manipulate it correctly. With the same hand he could select it afterward correctly from a group either tactually or visually or could draw it. By contrast, if the object was placed in one hand and any of the above responses demanded from the other hand, the performance was usually incorrect.

We interpret the difficulties in responding to or using language on the left in a similar way. Postoperatively, the patient was severely, although transiently, aphasic at the time that he had a dense right hemiplegia. This places his speech area almost certainly in the left hemisphere. His difficulties in producing language with the left hand or in responding to verbal commands requiring the use of the left side can be interpreted as resulting from deconnection of the right hemisphere from the speech area. The patient correctly named objects placed in the right hand and wrote correctly with the right hand both spontaneously and to dictation. Verbal commands were performed correctly with the right hand. By contrast, he generally was incorrect in naming objects in the left hand and wrote incorrectly with the left hand both spontaneously and to dictation. Verbal commands were frequently performed incorrectly with the left hand.

While the early cases were described as being apraxic in the left arm, we feel that a more precise delineation is possible in our patient. The patient showed no difficulties of performance of complex movements as long as stimulus and response were limited to the same hemisphere. The left hand showed difficulties only when the information had to be transferred from the left hemisphere, for example, in performing verbal commands, or drawing or otherwise identifying with the left hand objects held in the right (concealed from vision). But as we have shown, the right

hand was equally poor in performing movements whose stimulus came from the right hemisphere, for example, in drawing objects held in the left hand. In other words, apparently apraxic difficulties could be induced in either hand.

Where is the lesion producing this disturbance? The interpretation that this functional deconnection of the 2 cerebral hemispheres is a result of damage to the corpus callosum appears to us to be the most likely one. It is in harmony with the earlier anatomical observations and is concordant with the evidence that puts a lesion into the distribution of the anterior cerebral artery. It is consistent with the observations of Sperry (1961) and his students on the physiological functions of this commissure.

Sperry (1961) used transfer of learning technics to show callosal deconnection. Such experiments were not possible in our patient. However, our evidence of lack of interhemispheric transfer is as direct as that which Sperry obtained by use of learning technics. In fact, certain of our experiments would be difficult to do with animals, for example, drawing with one hand an object held with the other. Similarly, experiments on transfer of language are possible only in man.

It is difficult to be certain of the pathological nature of the involvement. At first, infiltration by tumor would appear to be most likely. However, we do not know whether any of the findings of deconnection were present before operation. Furthermore, there has been steady improvement over the six months since operation in the right hemiplegia and grasp reflex and in the signs of deconnection. This would appear to us to be too long to be accounted for by surgical trauma or edema. The patient underwent an extensive frontal lobe resection and some branches of the anterior cerebral artery were ligated. We think it possible that the vessel supplying the corpus callosum on the left was either tied off or developed a thrombosis in response to surgical trauma. No postoperative angiogram was done so that we cannot at this time affirm the correctness of this hypothesis.[1]

The evidence also suggests that the lesion does not involve the posterior callosum. The patient read words tachistoscopically presented in either visual field as quickly as normals. It is interesting to compare the patient of Trescher and Ford (1937), the posterior end of whose callosum had been sectioned surgically. The patient could not read letters in the left

visual field. In addition she could not name letters placed in the left hand but could name objects. The authors regarded this as a specialized tactile agnosia rather than as astereognosis on the basis of the excellent preservation of two-point discrimination and position sense and the dissociation between letters and objects. We feel it likely that if nonverbal identification of letters by the left hand had been tested, these authors would have found it intact and would have been able to strengthen their argument. The detailed differences between anterior and posterior callosal lesions will not be dealt with extensively at this point. This problem is dealt with more fully elsewhere (Geschwind, 1962).

The one feature of the case that may at first appear to be somewhat perplexing is the retained ability of the patient to perform certain learned activities that require the cooperation of the two hands, such as threading a needle. It can readily be demonstrated, however, that 2 normals, one using the right hand and the other using the left, can combine to perform successfully such activities. It seems obvious that each hemisphere of the patient had learned its role in such tasks, and that if 2 normals can cooperate in performing an activity, 2 isolated cerebral hemispheres can do so equally well.

The most serious objection to our interpretation is the negative results obtained by Akelaitis (1944) and his co-workers in their studies of humans with callosal sections. We are not certain of the reasons for these discrepancies but would suggest certain possibilities. Most, although not all, of Akelaitis' patients were young and had epilepsy dating from childhood. It is possible that early hemispheric lesions inhibit the development of unilateral dominance for language. Second, youth alone may be important – just as the young compensate for cerebellar removals while adults do not, it is possible that alternative pathways may be more readily available or more easily developed in the younger patient with a callosal section. It may also be significant that Akelaitis' patients, with only one exception, were epileptics of some years' duration. It is conceivable that the occurrence of multiple seizures might facilitate the availability of alternative pathways. Another possibility is that partial deconnection may be more seriously disturbing in man than total separation. Further, it is possible that only a small number of humans are as heavily dependent on the callosum as are lower animals, so that the syndrome might be seen only in such subjects. Finally, it should be considered that, in the presence

of intellectual deterioration, the patient is somehow less able to compensate for the destruction of the callosum (just as denial of hemiplegia in right parietal syndromes is more likely to occur in the presence of intellectual impairment). We cannot with assurance support any of these hypotheses.

Much of the early physiological work on the corpus callosum received its impetus from clinical observations. The physiological studies by Sperry and his co-workers are now in turn acting as a stimulus to clinicians to reinvestigate this problem. Human material offers certain special advantages, particularly the unilaterality of the speech area, that permit studies to be done that are not open to the animal investigator. We hope that further clinical researches in man will help to clarify some of these problems.

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NOTES

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Portions of the results were presented at the Boston Society of Psychiatry and Neurology on December 14, 1961.

[1] The patient died on June 3, 1962 (after the submission of this paper). Dr. José Segarra, chief of neuropathology at the Boston Veterans Administration Hospital, examined the brain grossly on June 21, 1962, and found evidence which supports the validity of the hypothesis suggested in the text. There was massive involvement of the left hemisphere by tumor which had extended downward into the basal ganglia, but no tumor was present on the right side. The corpus callosum showed no infiltration by tumor but was, on the contrary, markedly thinned in its anterior twothirds. The superior and medial surface of the left hemisphere extending back to the paracentral lobule was completely infarcted. The callosal and hemispheric lesions corresponded to the territory of the anterior cerebral artery.

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CHAPTER IV

CARL WERNICKE, THE BRESLAU SCHOOL AND THE HISTORY OF APHASIA

PREFACE

This and the next chapter represented my first ventures into medical history, as did another paper which appears later in this volume, 'Wernicke's contribution to the study of aphasia'. I did not then nor do I now consider myself a medical historian, or a historian or philosopher of science. It seemed to me, however, even as an amateur that it was important to apply some corrective views to what seemed to me to be grossly incorrect views as to the historical events, and as to the importance of certain key figures. Wernicke, like Liepmann, was a neglected figure, and Goldstein widely misinterpreted, a fact for which he was himself in good part responsible.

I also realized that the philosopher of science or the historian of ideas is often in a difficult position, like the anthropologist in a strange culture. His informant may wish to interpret his own scientific findings as supporting philosophical views to which his studies are in some instances either irrelevant or even contradictory.

I might point out that no adequate history yet exists in English, or very likely in any language, of the development of ideas in the higher functions. Furthermore the findings of this field have not had their proper impact upon the philosophy of science. One reason for this probably has been that while at least some writers on the philosophy of science have been well-versed in physics or mathematics, those who have written on the philosophical implications of disorders of the higher functions have always got their information from others. As my paper on Goldstein shows it is easy to see why they might often have been misled.

CARL WERNICKE, THE BRESLAU SCHOOL AND THE HISTORY OF APHASIA*

1963

The political difficulties of Germany at the close of the eighteenth century were reflected in the fortunes of the German universities, a large number of which either disappeared or merged with other institutions. The list of the universities affected included some of the most distinguished of the medieval foundations. Wittenberg, so closely associated with Luther and the origins of the Reformation, dissolved to be united with Halle in 1815. It was not alone. The two decades from 1798 to 1818 also witnessed the total or partial dissolution of the universities of Mainz, Cologne, Bamberg, Dillingen, Duisburg, Rinteln, Helmstedt, Salzburg, Erfurt, Altdorf, Frankfurt-an-der-Oder, Ingolstadt, and Münster.

It might have been expected that this massive disappearance of ancient seats of learning would have weakened seriously the position of German scholarship. The political conditions of the time might appear at first view to have been equally unfavorable to the flowering of learning. After the Congress of Vienna, Germany consisted of thirty-five princely states and four free cities, each in total control of its own internal affairs.

As is so often the case, however, the obvious rational expectation turns out to be incorrect, and the 19th century was in fact a period of great success for German scholarship. There were several factors which played a role in this upsurge of learning. For one, periods of confusion are sometimes more conductive to that passion and freedom which is so essential to great scholarship than are periods of undisturbed tranquillity. The professor who lives under the threats of major disturbances consequent on political upheaval may well feel it worth his while to be daring in the precious periods available to him for research and may not see the use of those cautious attempts to climb the academic ladder that are more likely to take precedence in periods of relative quiet. The many German scholars who made their positions even more untenable by their participation in forbidden political activities must also frequently have paid little attention to the long-range effects of their scholarly works on their formal academic careers.

But there were perhaps deeper reasons for the growth of the German universities despite the buffetings of the time. Reforms are often easier in periods of upheaval, when ancient practices lose their emotional force. Many of the universities which disappeared in the early 1800's were in fact, despite their illustrious histories, only medieval relics which deserved to vanish. Their dissolution in some cases strengthened other institutions which absorbed their faculties. The place of these extinct institutions was taken by newer and more vigorous centers more suited to the new times. Two of the greatest universities, Berlin and Bonn, were founded in the second decade of the 19th century, while Munich, another great seat of learning, was reorganized.

The crazy-quilt pattern of independent principalities probably was favorable rather than detrimental to the growth of learning. In such countries as France and England, where the medieval fractionation of power among independent duchies and baronies had long since been abolished, there had been a powerful trend toward centralization of cultural life in or near the capital, to the detriment of provincial cities and universities. In Germany, by contrast, almost any professor could feel satisfied that he was in or near a capital. Julius Caesar is said to have asserted that it was better to be first in some remote village than second in Rome. How much better, however, to be first in some remote village if it is also the seat of a king. Another benefit of the decentralization of power was that many states took pride in their universities and supported them more handsomely than would a central government anxious to concentrate its efforts on the aggrandizement of the capital. Many of the German princes prided themselves on their roles as patrons of learning.

There was still one further element in German university life which was beneficial to the widespread development of scholarship. One of the most useful features of medieval German university life had been carried over into the 19th century despite the decline of so many of the outworn traditions. The German student (often the able and ambitious one, although all too frequently the one whose main qualification was wealth) was likely to spend time at several universities, wandering like his medieval forbear to sit at the feet of great masters of learning. The professor who could build a metaphorical better mousetrap did indeed find the wandering scholars beating a path to his door. The great scholar or scientist could thus build his own prestige and not have to depend on that of his institution. This

system also favored an interchange of ideas that kept intellectual life in ferment. Even if the German professor was perhaps too often the supremely remote *Geheimrat* who brooked no disagreement, the tendency for the student to go elsewhere insured the infusion of new ideas into the system. Moreover, there was a strong tradition of appointment of professors from the outside, which tended to prevent departments from freezing into a uniform pattern. This tendency to appoint outsiders extended even beyond national boundaries to include all of the German-speaking world and even some non-German speaking countries where German scholarship was dominant. The orbit thus included not only the German states but also much of the Austrian empire and Switzerland, and even Holland.

German neurology and psychiatry, like the rest of German medicine, shared in the benefits of this system. It received tremendous impetus from the growth of nationalism, yet enjoyed the psychological and material advantages of decentralization, and so participated in the cross-fertilization resulting from the free movement of scholars. While in France and England the growth of neurology and psychiatry was very closely tied to London and Paris, these fields of knowledge flowered widely throughout the German-speaking world. The independence of the many great centers was so well established that it survived even the unification of Germany and the increased emphasis on Berlin as the capital of the amalgamated empire. This impetus carried well into the 20th century. Permanent major contributions came from such widely dispersed centers as Berlin, Breslau, Frankfurt-am-Main, Graz, Halle, Heidelberg, Jena, Königsberg, Leipzig, Munich, Prague, Vienna, and Zürich, and from such workers as Westphal, Jolly, Bonhoeffer, Wernicke, Alzheimer, Liepmann, Oppenheim, Edinger, Goldstein, Kleist, Krafft-Ebing, Wagner von Jauregg, Anton, Freud, Hitzig, Kraepelin, Nissl, Berger, Flechsig, Meynert, von Gudden, Grashey, Pick, Forel, Bleuler, Foerster, von Monakow, Vogt, Brodmann and Spielmeyer, to name only the most distinguished of those working at the turn of the century or immediately afterwards.

Aphasia was one of the most vigorously pursued areas of neurological study in the forty years from 1874 to 1914, and its fountainhead was Carl Wernicke, working in one of the most peripheral of the German universities, Breslau. This, the eighth largest of the German cities and a major industrial center, was the capital of the Prussian province of lower

Silesia. It must have been very different from the great universities of the German heartland because of its nearness to and long historical associations with Slavic Europe. It bordered on the Polish province of Galicia, which itself had a very heavy admixture of Jews on a background of Poles and other Slavs.

Carl Wernicke was born in Silesia, in Tarnowitz (now Tarnowskie Gory), not far from the Galician border and now also part of Poland. He grew up in a family of very reduced circumstances, worsened by his father's death when Wernicke was 17. His mother was nevertheless able to provide for his further education in the hope of his becoming a minister. Just before his final examinations, she too died. In 1870, at the age of 22, Wernicke was graduated in medicine. After a brief period in the university's eye clinic and as assistant to a surgeon in the Franco-Prussian war, he joined the psychiatric service at Breslau.

In 1874, when only 26, he published his epoch-making work on aphasia, *Der aphasische Symptomencomplex*, subtitled *Eine psychologische Studie auf anatomischer Basis* (Wernicke, 1874). This 72-page monograph was to set the tone for research in aphasia over the next forty years. Aphasic disorders had been recognized sporadically over several centuries and there had even been some impressive clinical studies; Professor Arthur Benton (1964) has reviewed these early developments. The modern history of aphasia clearly begins with Paul Broca, whose achievements have recently been reviewed by Macdonald Critchley (1961) and Robert Joynt (1964). There has been considerable argument between the supporters of Broca and those of Dax over the difficult question of priority. However, it appears that the work of Dax, like that of the other forerunners, was at best an isolated flash of insight which set no new activities in motion.

Why was Wernicke's work so important? He was certainly not the first to study aphasia after Broca. Hughlings Jackson indeed had already entered this field actively by the mid-1860's. Credit is often given to Wernicke for the discovery of those forms of aphasia in which comprehension is impaired. Yet Bastian had described such disturbances in 1869, as had Schmidt in 1871. Nevertheless, it was Wernicke's paper, not the observations of Bastian and Schmidt, which brought home to the neurological world the existence of the sensory varieties of aphasia. Furthermore, Wernicke's publication also succeeded in showing with postmortem material that this type of aphasia had a different localization from that

described by Broca. Thus he resolved the confusion that had followed on the finding of aphasic patients without lesions in Broca's area. Yet the most important contribution in this paper lay at a still deeper level: it is reflected in Wernicke's subtitle, 'A Psychological Study on an Anatomical Basis'. It is worthwhile to consider the previous history of aphasia in order to understand this point fully.

Broca's discovery resulted from an attempt to test a prediction made by workers in the phrenological tradition. It might have been anticipated that his success would have strengthened the cause of phrenology; actually, it hastened the downfall of that system since it increased the demand of neurologists for a theory which could link the new knowledge meaningfully with other knowledge – something phrenology, even in its most sophisticated form, could not supply. It was precisely this demand that Wernicke satisfied. Not only did he provide new evidence for the localization of aphasia but also set forth a theory which tied these phenomena to existing neurological knowledge. He thus made possible the development of a scientific approach to aphasia. On the basis of this theory it was possible to predict the existence of syndromes not previously seen and to devise experimental means of testing hypotheses. No one had previously supplied this. Little wonder that the appearance of this paper by an unknown 26-year-old physician, with less than four years of clinical experience and only six months study of anatomy under Meynert in Vienna, was considered so astonishing.

It was the theoretical aspect of Wernicke's approach that made it so fruitful over the next forty years and gave it such viability. However brilliant the clinical insights of a Jackson or a Head may have been, they suffered from not being readily derivable from anatomical facts nor being fruitful in devising new experiments. The Wernicke approach has been forced to adapt itself repeatedly to criticism, but in the end the core of Wernicke's method has remained consistently useful over the years.

Wernicke's reasoning was simple. He applied Meynert's teaching on the fiber tracts of the brain to the problem of aphasia. The phrenologists, he argued, had been wrong in their attempt to localize such complex mental attributes as magnanimity or filial love; what was actually localizable were much simpler perceptual and motor functions. All the complex array of human intellectual attributes must somehow be woven from these few threads of different texture. The cortex could at its simplest provide two

means of achieving this higher integration: it could store sensory traces in cells for long periods of time and, by means of association fiber tracts, it could link together different parts of the system. Meynert had already pointed out that what lay anterior to the fissure of Rolando was motor in function, what lay behind it was sensory. It seemed most reasonable to assume that traces of sensory impressions or of motor patterns should somehow be stored in regions adjacent to the appropriate elementary zones in the cortex.

The application to speech was immediate. Hitzig had already shown that at the lower end of the Rolandic cortex was a zone which, when stimulated on one side, led to bilateral movements of the mouth and the tongue. It was reasonable to assume that immediately in front of this zone lay a region where patterns of articulatory movements might be stored. This was exactly where Broca had placed the lesions in his cases, a localization repeatedly to be confirmed.

Meynert had asserted that the central end of the acoustic pathways lay in the vicinity of the Sylvian fissure. Thus it was reasonable to assume that traces of words should be stored near this zone. If this were the case, then an aphasia with loss of comprehension should result from lesions in this neighborhood. Necropsy of the patients recorded in Wernicke's paper amply confirmed these hypotheses.

Wernicke went further. The fact that these patients also showed an inability to comprehend written language was attributed to the circumstance that script is taught as an association to sounds and should therefore suffer with impairment in understanding sounds. Writing is lost for the same reason. The ability to repeat would obviously be lost. The speech of the patient would be fluent, even rapid and paraphasic[1] because of the loss of the internal correction of the activities of the motor speech zone by the receptive speech zone.

He argued further that a different lesion, sparing the receptive and motor speech zones but destroying the pathway connecting them, should lead to a characteristic syndrome, that of paraphasic speech (again, because of loss of the internal correction of the motor speech area by the receptive speech zone), and yet normal comprehension. He thought the lesions would lie in the insula.

This he called *Leitungsaphasie* or conduction aphasia. A severe critic of his own work, Wernicke was haunted by the problem of conduction apha-

sia over the next thirty years, since it seemed to him that the clinical pictures and anatomical findings he had expected did not seem to concord. Although he saw many patients with paraphasic speech and good comprehension, he did not always find the expected lesions. Further, he had seen lesions of the insula unaccompanied by this clinical picture. This confusing situation became clarified in two ways. Wernicke had first placed the pathway between the posterior and anterior speech regions in the insula, but he was later to be convinced by von Monakow's researches that the major pathway between the two speech areas lay in the arcuate (or superior longitudinal) fasciculus in the lower parietal region. Most later students of the problem have agreed with this localization.

There was, however, an even more important source of error which resulted from Wernicke having failed to analyze his own diagram correctly: he had omitted the deduction that there should be a disturbance of repetition in conduction aphasia. Lichtheim in 1885 correctly added the deduction and cited a patient showing this condition (Lichtheim, 1885). Freud returned to this problem in 1891 and, although he cited Lichtheim extensively, he seemed to have missed in part some of Lichtheim's extension of Wernicke's theory (Freud, 1953). Freud argued, as Lichtheim had, that the lesion disconnecting the motor from the sensory speech area should produce a loss of repetition in the face of intact comprehension, and went on to remark that this situation is highly unlikely. Yet within the next twenty years the triad of paraphasia, intact comprehension and impaired repetition was to become well known. In 1904 Karl Kleist, then an assistant of Wernicke's at Halle, demonstrated a case which finally convinced Wernicke of the existence of this entity. Kleist was to describe several such cases. In addition, the syndrome was described by others, such as Liepmann, Pappenheim, Pötzl, and Stengel. Kurt Goldstein (1927) (who had also been a student under Wernicke) called the complex of paraphasia with intact comprehension and poor repetition 'central aphasia'.[2] In recent years interest has again revived in this entity and its existence has been reconfirmed.

Wernicke's mode of analysis was quickly taken up. Lichtheim (1885) was able to predict the lesion necessary to produce pure word-deafness. It remained for Liepmann (1898, 1902) to conform this hypothesis by a necropsy on a superbly studied patient.[3] Dejerine's brilliant analysis of

isolated acquired reading defects was developed on a further extension of Wernicke's original analysis (Dejerine, 1892).

The final reason for Wernicke's importance extends beyond his work on aphasia. He had the ability, not common to all distinguished figures, of developing great students. The roster of those who came under his influence at Breslau is impressive: it was not merely that his students were distinguished; they learned to develop to full advantage that combination of talents which had served Wernicke so well, a profound respect for careful observation and a feeling for the elucidation of mechanisms.

His own activities extended into many fields and most of his contributions are now part of the foundations of neurology. He elucidated (with Mann, cf. Kirchhoff, 1921, 1922) the pattern of weakness in spastic hemiplegias, described what is now known as Wernicke's disease and its pathology, proved the origin of cortical deafness in bilateral temporal lesions, traced the pyramidal tract to the spinal cord, and established much of the picture of cortical sensory loss. He contributed greatly to developing the symptom picture of organic psychoses. He brought to recognition the important distinction between remote memory and the ability to learn new material.

This great productivity had a profound effect on his pupils. Karl Bonhoeffer was the first of Wernicke's assistants to be offered the 'Habilitation' (the first step in being formally permitted to teach publicly at a university). This followed Bonhoeffer's demonstration that a patient with unilateral chorea (until then always regarded as a functional psychiatric disorder) had a lesion in the midbrain. Bonhoeffer became professor in Breslau after Wernicke's departure for Halle, and subsequently was to hold the chair of psychiatry and neurology in Berlin. He devoted himself to many problems, including aphasia and cerebellar speech disturbances; he wrote the classical descriptions of alcoholic delirium and other alcoholic psychoses and of the memory disturbances in Korsakoff's syndrome; he delineated the generally accepted clinical manifestations of the toxic psychoses.

If the leading academic psychiatrist of Germany after World War I was a pupil of Wernicke's, so was the leading German neurosurgeon, Otfrid Foerster, who eventually became chief of the neurosurgical clinic in Breslau. In a series of papers (cf. Wartenberg, 1953; Zülch, 1956) published over a period of nearly forty years he recorded an impressive list of

contributions. He pioneered in the field of rehabilitation, made extensive studies of disturbances of movement and coordination and of the surgical treatment of these disorders. His work on sensation is classical and includes studies on the anatomy of the sensory pathways and on the neurosurgical treatment of pain. Some of his greatest contributions came from his studies of stimulation of the human brain and of epilepsy and its surgical treatment. He wrote little on aphasia but even this little reflected the influence of Wernicke's teaching. For example, the first description of the speech disturbances resulting from frontal parasagittal excisions was presented by Schwab and Foerster in 1926 (Schwab, 1926).

While Bonhoeffer and Foerster were the most prominent of his pupils, other students of Wernicke also made great advances, especially in the study of the aphasias and related disorders. His assistant Lissauer was to apply the same principles to an analysis of the study of the agnosias. Hugo Liepmann, another assistant, produced almost singlehandedly in a few years the classical clinical pictures and methods of analyses of the apraxias. Of all his students Liepmann brought Wernicke's method of analysis of the higher functions to its most brilliant and most significant development; his name in this field deserves a place with those of Broca and Wernicke.

Liepmann's early background contrasted sharply with that of Wernicke. While the master had grown up in difficult circumstances, Liepmann was born in 1863 to wealth, and remained a man of independent means throughout his life. He had begun his career in philosophy with a Ph.D. thesis on the atomistic ideas of Leucippus and Democritus, and later on he published a work on Schopenhauer. Liepmann received his second degree, in medicine, in 1894 at the age of 31. Already five years older than Wernicke when the latter produced his early masterwork, Liepmann was not long in making up for lost time. In 1895 he published his first neurological work (Liepmann, 1895), a pioneer study on the experimental production of hallucinations – a neglected pathfinding work in a field in which interest has revived only since the last war. That same year he went to Breslau to spend his four most important formative years. In this period he made his study of pure word deafness and confirmed at postmortem examination Lichtheim's anatomical predictions. In 1900 he returned to Berlin and during the next decade wrote his series of papers on apraxia; he was the first to use this term in its modern sense. Liepmann's first

important paper in this area, the study of the *Regierungsrat*, was published in 1900 (Liepmann, 1900). His accomplishment was twofold: he began by demonstrating that a patient who at first appeared to be globally demented had in fact an unprecedented combination of remarkable disturbances; he then proceeded to predict, on the basis of the known anatomy of the cortex and its fiber pathways, the localization of the lesions. Two years after the publication of this paper and its predictions as to the underlying pathology, the patient died and the necropsy confirmed Liepmann's expectations. Wernicke was especially delighted with this work, as well he might be: it remains to this day the most brilliant example of the 'psychological analysis on an anatomical basis' which the master had first developed a quarter of a century earlier.

Liepmann continued his work in the following years. In 1907 he and Otto Maas described the clinical picture resulting from disconnection of the hemispheres by a lesion in the corpus callosum which was demonstrated at necropsy (Liepmann and Maas, 1907). Goldstein, also a former student of Wernicke's, was to confirm this a year later with another proven case (Goldstein, 1908). Other case reports followed, including one published by Bonhoeffer (1914). This work was generally neglected in the English-speaking world and only today are we rediscovering the callosal syndromes which were common knowledge to Liepmann's contemporaries.

It may seem to some that this account of the Breslau school and its approach to aphasia neglects the existence of other streams in the history of the study of the higher functions of the nervous system. It might appear that there were really two separate streams, that initiated by Wernicke, which emphasized localization, and that including Marie, Head, von Monakow, Goldstein, and Pick, which was anti-localizationist. It would take us too far afield from our discussion of the history of the Breslau school to treat this problem thoroughly. None of their critics brought evidence to discredit the major conclusions of Wernicke and his followers. Head's clinical description and localization of syntactic aphasia, for example, were the same that half a century earlier Wernicke had given to that form of aphasia which now bears his name. Head's verbal aphasia obviously corresponds to the classical Broca's aphasia and indeed was given the same localization. Similarly, Pierre Marie's localizations of aphasia were highly classical despite his criticism of earlier approaches.

As Liepmann was to point out, the list of the localizations of apraxic lesions advanced by the great Russian-Swiss neurologist von Monakow was identical with Liepmann's much older list. Finally, Goldstein, in his writings over forty years (including his 1948 book on language), supported the main conclusions of the Wernicke approach and has always praised Liepmann's epoch-making work (Goldstein, 1948, 1953b).

Why this apparent discrepancy with the common view of two opposed streams? I believe that the answer will be found in a closer examination of the European literature on aphasia prior to World War I, on reading which one soon realizes that there was no wholesale rejection of the Wernicke approach by any of the major figures. But if the Wernicke approach remained, as it were, the central theme of nearly all the later efforts, it was to be elaborated in different ways. Wernicke had placed the pivot in his subtitle, 'A Psychological Study on an Anatomical Basis'. In one direction the theory could lead to an excessive multiplication of specific centers, a neophrenological approach, so to speak. Such proliferation of *ad hoc* centers had been as far from Wernicke's mind in 1874 as it was from Liepmann's a generation later. The extreme mosaicist view suffers from the same shortcomings as the phrenological view: mechanisms become unimportant and the whole point of linking psychology and anatomy vanishes. This pole of extreme mosaicism was supported not by Wernicke or his great student Liepmann, but by others such as Jendrassik, Henschen and Kleist.

If the theory ran into danger from a tendency of some toward excessive parcellation, it also ran into difficulties from the tendency of others to overemphasize the psychological study of aphasia, an approach which in its extreme form denied the importance of anatomy, or argued that aphasia must be considered completely separately on the psychological and anatomical levels. Even this view, however, was generally built on the core of the Wernicke approach. Thus Goldstein, who is often regarded as the spearhead of the psychological orientation, actually supported the main anatomical tenets of the Wernicke school; indeed some of Goldstein's writings, for example on callosal syndromes (Geschwind, 1964; Goldstein, 1927), explicitly supported Liepmann's analysis, which was obviously in the classical Wernicke tradition. It is striking that advocates of precise localization were often criticized for speaking of the concept of 'center', yet this notion reappeared extensively in Goldstein's writings as the

'concept field' (*Begriffsfeld*). Goldstein hastened to add that by this he meant no localized region but an extensive area of the brain outside the speech region (which he divided classically into sensory and motor zones). Yet Wernicke himself had disavowed a concept center as a localized entity and used this term in essentially the same sense as Goldstein's later use of *Begriffsfeld.*

It is perhaps not surprising that the differences between views lessen when they are considered from our current vantage point. In the heat of scientific dispute it may be easily overlooked that investigators, in fact, agree far more than they disagree. The central theme of Wernicke's approach survived the extremist pressures, and actually the study of aphasia gained from the criticisms. The attacks by the holistically oriented forced a constant reappraisal of clinical criteria so that nonfocal phenomena could be distinguished from focal disturbances; although such advocates of localization as Charcot had stressed the effects of early experience, the holists had also asserted the importance of this factor; it was the localizer Bastian who was praised by Freud (1953) and Goldstein for emphasizing that differences in the clinical picture might reflect not the site but the intensity of damage – the holists, however, advanced this point more persistently. Writers like Goldstein, agreeing with such highly anatomically oriented workers as Niessl von Mayendorf, stressed the equipotentiality not of the whole brain, which was clearly untenable in any strict sense, but of the individual more or less localizable 'center'; it was the holist Goldstein with his emphasis on the flexibility of the brain who made the strongest attack on the problem of re-education in aphasia, yet it was the localizer Foerster, one of Wernicke's leading pupils, who had been the first to establish rehabilitation as a major neurological activity. The holists were more active in linking linguistics to aphasia, yet Pick, one of the pioneers in this approach, had been one of the first to support Wernicke's views on the localization of sensory aphasias. If the holists were excessively outspoken, they were certainly not unimportant and they left their permanent seal on our thinking about aphasia; they studied new areas, fostered therapy, and forced refinement of clinical observation and anatomy. They had a profound effect on the course of the stream but they did not establish a new channel.

The mosaicists were also useful. If their anatomy had no link to psychology, their researches could be used by others who were more oriented in

that direction. They made fundamental advances in our knowledge of the gross and fine anatomy of the cortex. If the holists had an aversion to discovering new syndromes, the mosaicists performed a valuable service in discovering remarkably isolated disturbances which cried out for explanation. It is perhaps comforting to consider that the activities of highly skilled and intelligent men are rarely totally wasted, even when wrong. The history of discovery in aphasia would have been poorer if the field had been diverted into either channel; its future history will be the richer if the many channels explored by those on either bank of the main stream are investigated further.

The Breslau school, so richly rewarded in its scientific activities, was not to find the outside world quite so satisfying. Inadequate formal recognition, indeed even active administrative persecution, and violent events harassed the careers of the major figures of this school. Wernicke was involved in difficulties from the start. Following the publication of his early masterwork in 1874 he left Breslau to work in Berlin at the Charité Hospital under Westphal. But within two years he was dismissed because, as Liepmann (1922) put it, "he came into conflict with the direction of the Charité in a nonscientific and nonofficial situation, which was intensified by Wernicke's obstinacy..." What the situation was I have not found recorded, but one rumor at least is that it concerned a woman. Wernicke went into private practice with no academic connection. A possible appointment at Heidelberg was lost when he fell ill for six months. He applied for an appointment at Dalldorf, the Berlin municipal mental hospital, but was turned down, it is said at the instigation of the great pathologist Virchow, who was a powerful force in medicine in Berlin and who would not forgive the battle at the Charité. Wernicke was never allowed to forget the Charité incident. Not only was Virchow angered, but also Althoff, the Ministerial Director who harassed Wernicke for the rest of his life.

Finally, in 1885 an appointment at Breslau was realized. Wernicke's old friend, Förster, head of the ophthalmic clinic, succeeded in obtaining a post for his brilliant former pupil despite the opposition of Althoff, who had succeeded in blocking all other routes of advance. The years at Breslau were to have a bitter end, however. Wernicke had used the municipal psychiatric hospital as the source of most of his clinical material. He had tried fruitlessly for some years to have a university clinic built but had repeatedly been refused on economic grounds. Liepmann

(1922) records his repeated complaint, only too familiar to those in academic life, that "twelve million (marks) are granted without any further question for a warship, but the paltry million for a clinic is not to be had." The city fathers of Breslau, who had disliked the arrangement from the start, finally terminated the hospital connection; the psychiatric clinic now existed only on paper. For the next two years Wernicke was permitted to use patients for demonstrations in clinical lectures to students, but even this privilege was later withdrawn. Wernicke still had access to a neurological clinic and a private clinic. At least the marked decline in the work load of the department had the advantage of giving him considerably more free time to finish his *Foundations of Psychiatry* (Wernicke, 1906). It seems clear, however, that he lacked the ability to convince himself of the benefits of adversity.

Wernicke had more to suffer in these years. Three major professorships fell vacant. According to some he refused the chair vacated by Krafft-Ebing in Vienna in 1902. Niessl von Mayendorf claims that Wernicke would have been delighted to take over the chair once held by Meynert but that before he was approached several Viennese newspapers carried the statement that Wernicke would refuse if offered the position; according to von Mayendorf the rumor became generally accepted and therefore Wernicke never actually received the offer. A vacancy was created at Munich by the retirement of Bumm but was filled by Kraepelin, Wernicke's great opponent in purely psychiatric matters. Finally, the death of Jolly made available the directorship of the University Nerve Clinic at the Charité in Berlin, which Wernicke had left so unceremoniously twenty-eight years before. The medical faculty of the university nominated Wernicke as their first choice for the chair. But the Ministry of Education, under the direction of Wernicke's life-long antagonist Althoff, turned him down and Ziehen, the professor at Halle, was chosen instead.

In 1903 the direction of the Breslau psychiatric hospital was returned to Wernicke. Five years of frustration could not but leave their mark, however, and in 1904 Wernicke accepted an offer from Halle for the position being vacated by Ziehen in his move to Berlin. He thus took over the chair founded by Hitzig, the originator of the experimental study of localization in the human brain. A brief period of slightly more than a year was then to be perhaps the happiest in Wernicke's life; at last he had achieved his desire for a university clinic.

He also developed other interests: he had never participated in sports because of his slight build but now took enthusiastically to mountain climbing, skating and cycling. On June 13, 1905 Wernicke and his assistant, Berthold Pfeifer, went cycling in the Thuringian forest. They met a wagon loaded with logs and as they cycled past it, Wernicke fell. A rear wheel of the wagon ran over his breastbone. He apparently surmised that his injury was fatal and told Pfeifer that he probably had a hemorrhage into the pericardium. He was taken to a nearby country inn. Concerned that the wagon drivers should not be held responsible, he insisted that the blame was only his; it is recorded that in a quiet moment during this period he said, in the terminology of his own psychiatric textbook, "I am dying as the result of autopsychic disorientation." Kleist interprets this as meaning incorrect assessment of his own ability (to ride a bicycle). He died of his injuries on June 17, 1905, at the age of 57. The advances in thoracic surgery to be made a few years later might have saved him.

Of Wernicke's important pupils, Bonhoeffer was perhaps the most different personally. He appears from the descriptions of his biographers and the accounts of those who worked with him to have had the classical Greek quality of measure in all things. His own career had none of the upsetting character of Wernicke's. He left Breslau in 1903 for Königsberg and within a year took over the chair at Heidelberg which Kraepelin had vacated to move to Munich. Althoff, Wernicke's old nemesis, was angered by this change and threatened that Bonhoeffer would never again receive an appointment at a Prussian university. But with Wernicke's departure from Breslau, Bonhoeffer was called to take over his chair in that city. Althoff could not interfere since he was no longer in office. In 1912 Bonhoeffer went to Berlin, where he stayed for the remainder of his life.

This early history of uninterrupted success did not test Bonhoeffer's mettle and it was not until the tragic rise of Nazism in Germany that his tremendous personal strength was demonstrated. The rise of Hitler disturbed the usually well-ordered life of a professorial neuropsychiatric unit. Bonhoeffer handled these problems, as he had all others, with Apollonian equanimity. Quadfasel, one of his assistants, had shown his dislike of the new regime and was imprisoned despite his East Prussian Protestant ancestry; Bonhoeffer with the help of others was able to obtain for Quadfasel the immunity of a diplomatic railroad car to leave the country. Bonhoeffer's difficulties as the leading psychiatrist of the capital city of the

Reich must have been prodigious but, as recorded by Stertz (1956), he dealt with these problems with sovereign calm. He never had a picture of Hitler in his office, and after the passage of the sterilization laws by the Nazis he never reported a patient as hereditarily defective. Yet to a remark by an assistant that he hated the Nazis he replied, "A psychiatrist does not hate, he understands." His moral fiber seems to have been conveyed to others of his family circle. Of these the most distinguished was his son, Pastor Dietrich Bonhoeffer, who was despite his youth one of the leading figures in German protestant theology. He was one of the leading conspirators, along with his brother-in-law Dohnyani, in a plot to overthrow Hitler; arrested and imprisoned, he refused under torture to give the names of his co-conspirators. He and his brother Klaus were killed within a month of the end of the war.

Somehow the elder Bonhoeffer survived it all. A son who had also come through this period became one of Germany's leading physical chemists. Karl Bonhoeffer lived to enjoy an eightieth birthday. The *Monatsschrift für Psychiatrie und Neurologie*, founded by Wernicke and Ziehen and edited by Bonhoeffer for many years, honered him with a special Jubilee issue.[4] The American Psychiatric Association elected him an honorary member. Bonhoeffer regarded this, as Jossmann (1949) pointed out, as a token of the revived internationality of science rather than as a personal honor. He had himself noticed the warning signs of the stroke of which he was to die on December 4, 1948. In the announcement of his death was the phrase, "Despite severe suffering, a happy life."

Of all of Wernicke's pupils, Otfrid Foerster was to have the most brilliant worldly success. He refused to leave Breslau but the world came to his door. The list of those who studied with him is a roll call of the modern leaders of neurosurgery. Sherrington even expressed the view that he was worthy of a Nobel prize. Personal tragedies, however, clouded his life. The coming of the Nazis added further burdens. His wife was of Jewish extraction and some of his outspanding assistants had been Jewish. In addition he was himself probably suspect for having spent two years in Russia as the physician of the hemiplegic and aphasic Lenin. Foerster lacked Bonhoeffer's Olympian detachment in the face of the Nazis and personally suffered more from the political changes. He died June 15, 1941 of tuberculosis.

Of all of Wernicke's assistants at Breslau, Liepmann is recorded by

Kleist as being closest to him, and it was certainly he who was to repeat most exactly the first brilliant achievement of the master. His career was much like Wernicke's. After leaving Breslau, he returned to Berlin to work at Dalldorf, where Wernicke had been refused a post many years earlier. For nearly all the remainder of his career he stayed there, never to be offered a professorial chair despite the wide recognition of his work and his acceptance in Berlin as one of the outstanding lights of German neuropsychiatry. He was Jewish and, as Max Weber pointed out, Jews could not expect to advance in German academic life. Goldstein (1953b) mentions that Liepmann was told he would be offered an academic position if he changed his name and adopted Protestantism; although not religious, he refused on principle. His later years were darkened by the onset of Parkinson's disease. On his sixtieth birthday the *Zeitschrift für die gesamte Neurologie und Psychiatrie* honored him (Isserlin, 1925). Two years later, faced with the relentless progression of his disease, he committed suicide. So died Wernicke's most direct heir, "a worthy representative," as Goldstein put it, "of a great creative epoch in neurology."

NOTES

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[1] The term 'paraphasic' is used to characterize particular incorrect word usages in the speech of aphasics. Paraphasias may consist in omission of parts of the word, substitution of incorrect sounds in the word, correct words incorrectly used, or complete neologisms.

[2] Some authors have recently used Goldstein's term 'central aphasia' in a much broader sense than Goldstein. He specifically equated his 'central aphasia' to conduction aphasia.

[3] Some authors mistakenly assume that Wernicke described pure word-deafness in his 1874 monograph. He did not and, as already noted, the aphasia he described involved all aspects of speech. Lichtheim predicted and Liepmann demonstrated that pure word-deafness was the result of disconnection of auditory stimulation from an intact Wernicke's area.

[4] *Mschr. Psychiat. Neurol.* **117** (1949) No. 4/5/6.

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CHAPTER V

THE PARADOXICAL POSITION OF KURT GOLDSTEIN IN THE HISTORY OF APHASIA*

1964

ABSTRACT. A widely-accepted version of the history of the study of aphasia states that before the First World War this discipline was dominated by a group of workers who, with occasional exceptions, were unsophisticated psychologically and who attempted on the basis of inadequate clinical and pathological data to fit aphasia into a narrow anatomical scheme. According to this view a group of reformers appeared, Marie, von Monakow, Head, and Goldstein, who destroyed this inadequate classical scheme.

In this paper this view is criticized, and it is pointed out that none of the reformers made as great a break with the past as is generally thought. The work of Kurt Goldstein is particularly discussed and it is pointed out that it is readily apparent from Goldstein's own works that he accepted a majority of the classical teachings, indeed even in details. It is also pointed out that many of Goldstein's holistic theoretical views were in fact so extensively qualified as to make them compatible with almost any approach. There is some discussion as to the reasons for the widespread rejection of the classical views when even its apparently severest critics accepted so much of the classical techings.

In concluding it is pointed out that Goldstein is indeed a great figure in the history of aphasia, but that his contribution must be reassessed as a brilliant extension of the works of his illustrious predecessors, not as a completely new approach based on the destruction of older unsound views.

Until approximately three years ago I had accepted what to a very great extent has become the 'standard' history of aphasia. Broca's achievement was seen through a distant mist. Following his arousal of interest in the field came, we are generally told, the long Dark Ages. For the next 60 years the field was apparently dominated by a group of scholars, mostly German, who studied aphasia entirely from an anatomical point of view. Indeed, the argument often goes, it was a study from a point of view of fictional anatomy, barely, if at all, above the level of the phrenologists who had only so recently been deposed. Only Hughlings Jackson, unrecognized outside his own country, was said to be free from the overriding neurological mythology.

According to this standard history by the mid-1920's the situation had changed and the classical school was in full flight. The battle had been led in France by Pierre Marie, in Switzerland by von Monakow, in Germany by Kurt Goldstein, and in England by Henry Head. The ideas,

or what were thought to be the ideas of this group, soon gained domination in the English-speaking world. Head (1926) had been shrewd enough to point out that much of the great German growth of neurology had been related to their victory in the Franco-Prussian war. He was not shrewd enough to apply this valuable historical lesson to his own time and to realize that perhaps the decline of the vigor and influence of German neurology was strongly related to the defeat of Germany in World War I and the shift of the center of gravity of intellectual life to the English-speaking world, rather than necessarily to any defects in the ideas of the German scholars.

Chaos, as is well known, was Head's word and if chaos was perhaps not present before Head, it was certainly fully evident after him. Somewhere about 1960 I awoke, perhaps belatedly, to my own profound confusion. I had regarded myself as a member of the philosophically sophisticated Jackson-Goldstein-Head group. Yet, I was persistently troubled by the fact that people who had left their mark so indelibly in many areas of neurology, such as Wernicke, Bastian, Dejerine, Charcot, and many others, could apparently have shown what was asserted to be the sheerest naivete and incompetence in the area of the higher functions. It seemed difficult to accept the view that men who had established long-honored clinical pictures should have apparently been so incapable of examining an aphasic, or that scholars who had made fundamental anatomical investigations of permanent worth should have been so perfunctory and sloppy in their descriptions of the brains of aphasics. I, along with several of my colleagues of the Aphasia Unit of the Boston V.A. Hospital, decided to reinvestigate the problem at its source. Rather than accept the authority of even so distinguished a group as Marie, von Monakow, Head, and Goldstein, we would go back to the original literature and see for ourselves whether indeed the evidence was so poor as had been suggested. This was no minor problem. Practically none of this literature was available in English, except occasionally in accounts by its detractors, and much of it is contained in old journals, many of which are no longer published, and often difficult to obtain. The picture that emerged from this study was not the classic one of the chaotic confabulations of a group of tedious imperceptive German scholars but rather that of the healthy active disagreements of a lively science. The broadside accusations of carelessness, of inadequate examination, of unconcern with psychology

were all unfounded, at least when the best of the classicists were involved. There certainly had been people in this era who had indiscriminately collected series of well and ill-described cases, but the best had never done so.

Actually, my colleagues and I were now even more confused. We had, in fact, expected to find that most of the criticisms of the great reformers would be justified and we were quite unprepared to find the case to be so clearly otherwise. It, therefore, became necessary in turn to go back to the writings of the leaders of the 'reform movement' in aphasia and to reread them as carefully as their predecessors.

Jackson, we rapidly found, did not represent a problem. His early disagreement with Broca was long since a dead issue. As for his later writings, we found that writers as diverse as Head (1926), Nielsen (1946), and Penfield and Roberts (1959) all quoted him with equal approval. Otto Sittig (1931) who was in major part responsible for the introduction of Jacksonian ideas into German-speaking neurology after World War I, quoted Jackson extensively and approvingly in his monograph on apraxia in which he also accepted the fundamental analyses and observations of Hugo Liepmann, one of the greatest of the classical localizationists.

Pierre Marie had inveighed against Dejerine in the early 1900's and had carried on a bitter battle against classical views. But when Marie and Foix (1917) published their diagram of the lesions producing different aphasic syndromes, it was little different in fact from the diagrams of Marie's predecessors. Foix, one of Marie's major pupils, certainly shows no anti-localizationist bias in most of his writings.

Von Monakow was another one of the reformers. Just before World War I, Hugo Liepmann (1914) wrote a long reply to von Monakow's chapter on apraxia (itself nearly 100 pages long) in which Liepmann had been seriously taken to task. Liepmann quotes page after page of gross misquotation and misunderstanding from von Monakow's work (having read Liepmann with care, I agree with his objections). But the unkindest cut of all for Liepmann was to find that after many pages of criticism, von Monakow himself came to the same conclusion! Thus, Liepmann writes, "Von Monakow... finally... arrives at his verification of all my propositions relating to localization without commenting that they are mine." He then quotes von Monakow's list of sites of predilection of

apraxia, which indeed do coincide with Liepmann's older list. The monograph on apraxia by Victoria (1938) based on von Monakow's work also comes to conclusions that had been clearly stated by Liepmann, although this author also is critical of Liepmann.

Henry Head (1926) is often remembered as a major reformer. Yet, after inveighing against the diagram makers and damning their diagrams, he ended his book with a very classical set of localizations for his four types of aphasia, each carefully provided with a new name so as effectively to obscure the fact that in most instances his findings coincided with those of earlier workers! Any reader who goes to the discussion of specific aphasic syndromes in Russell Brain's (1961) writings will find that for all of Brain's admiration for his teacher, this author clearly reveals a return to many of the older views which Head had so dearly wished to destroy. It is even remarkable to find that Head had substituted the word 'schema' for the hated word 'center' of some of the classical writers. Yet, the definition given by Bastian (1898), one of Head's favorite *bêtes noires*, for a 'center', is little different from Head's definition of a schema!

Thus Bastian wrote, "... There must be certain sets of structurally related cell and fibre mechanisms in the cortex... Such diffuse but functionally unified nervous networks may differ altogether from the common conception of a neatly defined 'centre', and yet for the sake of brevity it is convenient to retain this word and refer to such networks as so many 'centres'..."

I would not in what I have said so far wish to suggest that any of the authors I have mentioned were trivial or insignificant men, nor indeed that they did not contribute to the advance of knowledge of aphasia. I would only wish to suggest that they were not so revolutionary as they thought they were and that for all their protests they were nearer to the classical tradition (which was in any case not at all monolithic as is often suggested) than they asserted that they were.

To me, however, the most interesting of the four reformers is Kurt Goldstein, who has actually had much more profound effects on thought about the higher functions, certainly in the United States and probably even in England, than any of the others, even including Head. There are several reasons why Goldstein has been so influential. For one thing he has long outlived the others who have receded into the classical past and has continued to work long beyond them – Goldstein's recent book (Goldstein,

1948) although based heavily on his earlier German monograph (Goldstein, 1927) on localization in the cortex, was still a presentation of his ideas more than twenty years after Head's two volumes. Furthermore Goldstein's work in the twenties and thirties, after the other authors had left the scene, coincided with the great rise of holistic approaches in other fields, such as psychology, psychiatry, anthropology and sociology and therefore found an audience receptive, particularly in the United States, to its general approach. In addition, although the others, particularly Marie and Head had stressed what they liked to call the 'psychological' approach, Goldstein's work was much more closely tied to the academic psychology of his era in Germany and later in this country. His work has therefore come to be widely diffused to large numbers of undergraduate students of psychology in many American universities, a group generally poorly informed both as to the work of the other reformers and almost totally uninformed about the classical neurologists criticized by this group.

I was therefore most intrigued to review Goldstein's work in the light of the fact that he is often regarded as the greatest influence in the revolt against the classical school of thought about aphasia. I had, of course, read some time ago the monograph (Goldstein, 1948) on language disturbances but distrusted my memory of it. I had already been surprised in reading other writers, such as Lange (1936) and Isserlin (1936), that they frequently quoted Goldstein's opinions on matters of localization. I began by reading and later by re-reading the work on cerebral localization, published in 1927, in the peak period of Goldstein's productivity. The arrangement of this book is a familiar one. Most of the first half is devoted to a discussion of theoretical principles and at first glance sounds like what most of us have been accustomed to think of as Goldstein's views, critical of classical approaches and stressing organismic considerations.

Roughly the second half of the book constitutes the 'Special Part'. Goldstein opens this part by pointing out that it would be most desirable to deal with the syndromes of each cytoarchitectonic region. All through the Special Part he stresses the details of cytoarchitecture and indeed reference back to the theoretical section shows that even this section began with an extended presentation of cytoarchitecture and myelinogenesis and indeed a defense of the work of Flechsig.

We come next in the 'Special Part' to a long, elaborate, highly classical

discussion of the motor regions, with several citations to Goldstein's own observations, and certainly little or no intrusion of the considerations of the theoretical section. The following section is on the sensory regions, another superb but classical discussion. In this section he cites, for example, cases of isolated loss of sensation confined to the ends of the thumbs and says, "These circumscribed deficits correspond to circumscribed lesions in the posterior central gyrus." He even goes on to express the view that there is probably a detailed projection of the periphery on the sensory cortex. He took special care to refute the critics of such a projection, e.g., Bonhoeffer, a very classical neurologist.

The next section is on the visual system, again highly classical. Anyone who thinks that Goldstein did not believe in isolated defects should note his comment that "*we* have confirmed many times" what Liepmann occasionally observed, that isolated disturbances of color sense without visual field defect can occur particularly with lesions in the left hemisphere. His discussion of what he calls the first form of psychic blindness states that, "There is often bilateral disease of the occipital lobe. (If there is a unilateral lesion it lies)... always in the *left* hemisphere (in right-handers) and indeed it then involves either the calcarine cortex or the optic radiation and the callosal fibers, so that excitations from neither the right nor the left half of the retina can get to the left hemisphere..." This is, needless to say, a highly classical connectionistic interpretation. Goldstein then goes on to dismiss the idea that this disorder is the result of the field defects; he rejects Niessl von Mayendorf's assertion that psychic blindness is the result of lesions of the macular bundle bilaterally. He then says, "The simultaneous occurrence of diffuse cerebral damage, as von Monakow assumes, is *not* necessary (for this clinical picture). There can persist a lasting psychic blindness even in an (otherwise) intact youthful brain with a quite circumscribed lesion..." I am not myself sure that this view is necessarily correct but I would hardly assume that it could be considered revolutionary by classical localizationists.

We turn to the section on aphasia, where perhaps we might expect a more 'Goldsteinian' approach. At first it appears that we are about to break totally with the classicists. He writes "...in our opinion these (distinct forms of aphasia) do not exist in reality..." Yet, two pages later we find that this is not the case. (Let me stress that these quotations from Goldstein are not 'taken out of context'. I have in each instance been as

careful as I could be to ascertain that these were Goldstein's personal views.) He writes of the "symptom-complex which is usually characterized as word-dumbness or subcortical aphasia and which I would characterize as the peripheral form of motor aphasia..." He notes that writing may be normal, that reading is undisturbed and that the anatomical substrate is a lesion of the classic Broca's region. He argues that this region is slightly wider than the usual classical zone, but concludes that the pars triangularis and orbitalis of the third frontal convolution do not belong to this region. He disagrees with Pierre Marie's view that lesions of this region produce dysarthria and not aphasia, and goes on to agree with Niessl von Mayendorf's inclusion of the Rolandic operculum in the anterior speech area although he has some disagreements with him. He goes on to say that by "an exclusive lesion of F_3 with intactness of the central operculum, severe motor aphasia, even persistent, can appear..." Any of this could have been written by the staunchest of the classicists.

He does mention in this section that the presence or absence of writing defect with a Broca's aphasia is not only a question of anatomy but may be dependent on whether the patient had always been accustomed to speak when writing or not. In fact, however, this type of 'functional' explanation had already been widely used by such classical localizers as Charcot. Goldstein goes on a page or two later to attribute restitution of function in some cases of Broca's aphasia to the taking over by the right side of some of the functions previously carried on by the left.

I need not pile on the examples in too much detail. He speaks a few pages later of the transcortical aphasias and refers to his own book on this topic which is certainly rich in anatomical discussion. He goes on to discuss pure word-deafness and agrees with Liepmann's views on the anatomical mechanism of this disturbance. And so it continues for page after page.

Perhaps it will be argued that his views had not yet fully matured and that he had as yet not absorbed fully his own philosophy, as expressed so forcefully in his introduction to the 1927 monograph. Let us therefore turn to the Goldstein of 1948. Again much of the book is taken up with theory and indeed with theory of the type that we would expect. Even so, we find him saying on page 47, "...the assumption of different functions for the different layers of the cortex is most probably justified." On page

55 he refers to earlier work of his own (published first in 1908) which showed that "tactile agnosia in the 'minor' hand may result from a lesion of the corpus callosum." On page 82 we discover that "It was Bastian who introduced the functional factor into the theory of aphasia for the explanation of the different preservation of different performances..." But this is the Bastian whom Head had criticized as one of the worst of the classicists! On page 129 we read the astonishing statement that "the collection of cases by Henschen leaves no doubt about the significance (of the second frontal gyrus) for the development of this particular form of agraphia..." It is perhaps surprising to see Goldstein agreeing with Henschen when so confirmed as localizer as Sir Charles Symonds (1953) finds the evidence for pure agraphia poor! On the next page Goldstein writes, "My case leaves no doubt that the anatomic cause of (agraphia of the minor hand) is a destruction of the corpus callosum as Liepmann first showed..." By the time we get to the second half of this book we find most of the 1927 conclusions repeated. Certainly there are more holistic discussions in the text but without question there are innumerable areas of agreement with the classical neurologists.

Must we then ask, 'Which is the real Kurt Goldstein?' I frequently find that when I quote Goldstein on localization someone asks me, 'Do you mean *Kurt* Goldstein?' One man protested that he had read Goldstein's books and never seen any of the things that I had mentioned. I asked whether he had read the second half of the 1948 book on language disturbances, in which the case records and the discussion of individual syndromes were concentrated. He admitted that he hadn't on the assumption that it was merely 'technical detail'. I am certain that like this man when I read this book for the first time I had omitted or glossed over the 'technical detail'.

Actually the discrepancy between the two Goldsteins is less great than one might assume. Careful re-reading of the theoretical section in the 1927 monograph shows that many of the 'philosophical' principles are followed by so many qualifications that they need not conflict with any theory. Thus, Goldstein writes, "Since in the organism one is dealing with a unitary system, a stimulus would have to manifest itself everywhere in an alteration of the same kind." But a paragraph later he goes on to say, "Usually the effect of the change following on an adequate stimulus does not show itself everywhere, but especially in a more or less circumscribed

region of the body..." He gives the reasons for this. The first is, "The alteration in response to a stimulus in a large system does not occur everywhere simultaneously and with equal intensity, but occurs sooner and more strongly near the site of the stimulus than in more distant regions..." Another reason he gives for differential responses is that "different parts of the nervous system are differently attuned to the different types of stimuli..." hardly a statement to disturb even the most die-hard of localizationists. A few pages later he states that "all forms of damage to the nervous system can be regarded from the point of view that one is dealing with an *isolation* of a certain part. All lesions of the nervous system exclude a certain part of the system." But this would suggest that a view of the organism as a unity might not be too useful.

What conclusion are we to draw? Kurt Goldstein was and is a great figure and a great contributor to the study of the higher functions of the nervous system. But his contribution was not totally to destroy his predecessors, to abolish the diagrams, or to bring order into the chaos. Few scientists create revolutions and the revolution in aphasia occurred in the 1860's with Broca and in the 1870's with Wernicke. Most people who advance their field must disagree with their predecessors to some extent and in some measure destroy the past; they must also disagree with their contemporaries and so increase chaos. Usually a field is least usefully active when it is apparently least chaotic.

Goldstein, like Head, von Monakow, and Marie, made advances and indeed great advances in the study of the higher functions. But rather than being the initiator of a totally new approach, he was a brilliant developer and reviser of a magnificent understructure developed in the years before his arrival on the scene. Aphasia did not stop in 1920 but it certainly did not begin at that time. What I have tried to show in part is that our standard view of the history of aphasia is at least a seriously distorted one. At its worst, this view of history has closed certain useful avenues of approach and forced us to forget important truths that even the so-called reformers accepted. The widespread neglect of the important elucidation of the functions of the corpus callosum by such people as Dejerine, Liepmann, and Goldstein himself at the turn of the century is an excellent example of the damage done by this failure to appreciate the past; significant experimental and clinical study was almost certainly delayed for many years by this neglect of the classical localizers.

How then are we to regard Goldstein's position? His contribution as a localizer in the classical sense is in fact highly significant although rarely taught. He also made a contribution in other ways. We should recall that his stress on looking at the background of the patient and at the intensity of the lesion were already stated by some of his classical precedessors. It should be noted that few of them were as excessively localizationist as is often assumed – in fact, some of them were less localizationist than Goldstein in relation to their interpretation of many issues! Goldstein stressed more than his predecessors the reaction of normal parts of the nervous system to illness in one portion and highlighted the importance of looking at more than the obvious impairment of the patient. But these and others of his so-called principles should not be regarded as great philosophic truths. It is far better to say that he suggested some ways of looking at disease of the nervous system which are sometimes useful. Thus was Goldstein correct in stressing the 'total response of the patient'? There is no yes or no answer. Instead, we should say: Some phenomena are clarified by regarding them from the point of view of organismic reactions, others are clarified by being regarded atomistically. The major source of error is the failure to look at each phenomenon from both points of view to see which is most appropriate.

Perhaps I could most appropriately close with a recent statement by Goldstein (1953) himself about Hugo Liepmann, Wernicke's great pupil, and probably the most brilliant of the classical localizers in his synthesis of anatomy and behavior, "Liepmann's work will remain one of the landmarks of our knowledge of the function of the brain. He is a worthy representative of a great creative epoch in neurology." Evidently Goldstein did not reject his predecessors as much as many other people thought he had.

NOTE

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CHAPTER VI

NON-APHASIC DISORDERS OF SPEECH

PREFACE

Edwin Weinstein (now at the Mount Sinai Medical School) was the first person to draw my attention to a whole array of remarkable phenomena which can readily be mistaken for primary disorders of memory or language. I should hasten to add that this should not suggest to the reader that Weinstein shares my interpretation of many of these phenomena, since there are many points on which he and I will continue to carry on a friendly argument for many years. Both this paper and the one later in the volume 'The varieties of naming errors' as well as my thinking on denial syndromes all reflect, however, my debt to Weinstein's highly original observations. These were, and perhaps still are, neglected by neurologists who thought that Weinstein was advancing a purely psychiatric explanation of the syndromes of denial and their accompanying errors in naming and distortions of memory. This belief was a naive misinterpretation of Weinstein's more refined argument, i.e., that a special type of motivated denial behavior is seen in patients with certain neurological disorders.

NON-APHASIC DISORDERS OF SPEECH*

1964

ABSTRACT. A review is presented of the non-aphasic speech disorders, i.e., those speech disorders which differ from those produced by lesions of the classic speech regions of the brain. Non-aphasic speech disorders include those seen in functional disease, those occurring under conditions of widespread (rather than focal) disturbance of the brain, and those which occur with focal epileptic disease outside the speech regions and particularly on the right side of the brain. Criteria are given for distinguishing aphasic from non-aphasic disorders and the practical and theoretical importance of these distinctions is emphasized.

The separation of aphasic from non-aphasic disorders of language has a significance which extends well beyond the simple desire of the scholar for neat ordering of his data. This distinction is of importance for the clinician who may in the presence of such disorders mistakenly assume the presence of disease of the classical speech regions in the left hemisphere and thus be led to an incorrect localization with resultant errors in investigation and management. Organic and psychiatric disease may be confused to the disadvantage of the patient. The physician may be misled by the malingerer to the disadvantage not of the patient himself but of those whom he is desirous of misleading.

The practical uses of this knowledge would alone justify calling it to the attention of physicians. To the researcher in the field of aphasia the question has an importance transcending practical application in the clinic. The mistaken identification of a non-aphasic disorder as aphasic can lead the investigator to many errors. He may be led to compare the behavior of patients who in fact are not similar. The student of clinico-anatomic correlations may be particularly confused by his post-mortem findings. Thus in some clinics cases of 'aphasia' without post-mortem lesions in the classical locations are frequently found. In at least two instances with which I am familiar these cases should have been either recognized or strongly suspected to be non-aphasic speech disorders.

Let us outline here the groups of patients who will be of concern to us. We wish to distinguish first those patients who manifest speech disturbances on a functional psychiatric basis from those with neurological

disease. Among those with neurological disease we wish to separate those with diffuse disturbances of the brain from those with focal abnormalities in the speech regions.[1] We will organize our material according to the clinical pictures presented by the patient.

Various aspects of the problem of non-aphasic speech disturbance have concerned students of aphasia since its beginnings. Thus Wernicke (1874) pointed out in his classic monograph the common error of confusing fluent paraphasic speech with the productions of psychotics. Critchley (1964) has recently discussed this problem. A problem of particular interest in classical neurology was that of anomia or amnesic aphasia. One group of neurologists regarded isolated naming difficulties as resulting from localized lesions in the left posterior parietal region (a view held for example by Head, 1926, despite his well-known prejudices against localization); while another group insisted that anomia was the result of diffuse disease. Some neurologists (e.g., Gloning, Gloning and Hoff, 1963) have supported the existence of both localized and unlocalized anomias. As will be shown later, it is likely that there exist both localized and unlocalized anomias but their clinical pictures differ. It is primarily to the work of Weinstein and his associates (Weinstein, 1958; Weinstein and Kahn, 1955; Weinstein and Keller, 1964) that we are indebted for the clarification of the syndrome of non-localizable anomia.

I. MUTISM: PATIENTS WHO SAY NOTHING

As I learned several years ago from Dr. Macdonald Critchley mutism is uncommon among aphasics, and the most limited aphasic usually has at least one utterance. The occasional aphasic who is mute usually exhibits certain other characteristics which are helpful clinically. Thus he nearly always attempts to communicate in some way, e.g., by gestures or grunts, although he may not succeed. His writing is usually impaired to roughly the same extent as his speech.

Patients with certain psychiatric syndromes may present with mutism. The patient mute as the result of depression will nearly always have a history of increasingly depressive behavior and will manifest many of the other characteristics of that disorder. Schizophrenia and hysteria may present with mutism. The age of the patient is a valuable aid here since the majority of the aphasias occur in an older age group while schizo-

phrenia and hysteria are disorders which almost always appear before the age of 30. Schizophrenic or hysterical mutism may well appear in patients who already have a long history of behavior disturbance while most aphasias tend to be of acute onset in people who have been behaviorally normal. Both the schizophrenic and the hysteric patient may write normally despite their mutism. Hysteria is less likely to present with mutism than with aphonia, i.e., a loss of voice. This type of patient (who whispers but produces correct language) should present no diagnostic problem.

There are also neurological causes of non-aphasic mutism. Thus mutism may reveal itself in cases of widespread involvement of the brain by the metabolic disturbances of medical illnesses, such as high fever, dehydration, uremia or congestive heart failure, in intoxications, or in widespread involvement of the nervous system itself, for example, diffuse dementing disorders.

In these cases (of dementia) the advent of mutism signifies not the involvement of the speech areas or their connections but rather that the dementing process has gone so far that in effect the patient has nothing more to say.

Mutism may also result from focal pathology. There is a small group of patients who present with the acute onset of a right hemiplegia and total mutism but with retained ability to write. Following Bastian (1898) we have designated these patients as aphemic rather than aphasic. The reason for calling this disorder non-aphasic is based on the fact that those patients we have seen with this syndrome have manifested no positive signs of aphasia as soon as there was some recovery of speech. With only slight recovery of speech these patients show not the usual picture of a Broca's aphasia with markedly telegraphic speech but rather markedly effortful speech without agrammatism or other aphasic features. A more common picture is that of the patient with bilateral lesions leading to a classic pseudo-bulbar dysarthria. This disturbance is, however, uncommonly so severe as to produce a total state of mutism. The ability to write may of course be totally preserved in this situation. The other obvious signs of pseudo-bulbar palsy make this diagnosis easy. Mutism may be seen in the patient with multiple lacunar infarcts in the basal ganglia. It is also seen sometimes in patients who have undergone thalamotomies, particularly after bilateral operations.

More challenging are those cases of mutism without pseudo-bulbar

signs or hemiplegias. These are the cases with show the syndrome of akinetic mutism, and their failure to speak is not isolated but is only one aspect of a general failure to act. Occasionally such patients will whisper faintly although they will not speak. They differ from the hysterical aphasic, however, in that it takes great effort to bring the akinetic mute to whisper weakly while the hysterical aphasic may be quite prepared to produce considerable speech in a loud whisper. The akinetic mute may be particularly confusing for several reasons. Occasionally he will not speak spontaneously, but will, like some mute schizophrenics show echolalia (this disturbance will be discussed later on in this paper). He may show a great variability in behavior, occasionally spontaneously producing a perfectly good sentence, and then lapsing back into mutism. In some instances he may show some spontaneous speech but this may manifest the non-aphasic naming disturbances to which we will return later. Akinetic mutism can occur in disease of the upper brain stem or may result from bilateral involvement of the frontal lobe anterior to the motor regions.

Mutism in children is a more difficult problem than in the adult. As we have noted acquired aphasias in the adult rarely lead to mutism but they do so commonly in the child. Even lesions which in the adult usually lead to fluent paraphasic speech, (i.e., temporal and/or parietal lesions) are likely to lead to mutism in childhood. The difficulty is further compounded by the fact that mutism tends to be more prominent as a manifestation of psychiatric disease in the child than it is in adults. In children extreme caution is needed in distinguishing the aphasic from the non-aphasic.[2]

II. ECHOLALIC DISTURBANCES

Stengel (1947) has written an excellent article on the functional characteristics of echolalia. Echolalia is rare in aphasias, at least in those aphasics with lesions in the classical Broca's or Wernicke's areas or their connections. It can occur in those syndromes which were called 'trans-cortical aphasias' by the classic neurologists. It was on this group of cases that Kurt Goldstein (1917) wrote an extensive and detailed monograph. By trans-cortical aphasias were meant a group of disturbances in which repetition was very well preserved in contrast to other disturbances in the production or comprehension of speech. The trans-cortical aphasias constitute a bridge between the aphasic and non-aphasic disorders of

organic origin. Thus trans-cortical motor aphasia (in which repetition is excellent despite a lack of spontaneous speech) was thought by Goldstein to imply a lesion lying anterior to an intact Broca's area. Such patients may be nearly or totally mute except in repetition of the examiner's speech. Trans-cortical sensory aphasia (in which repetition is excellent in the face of grossly impaired comprehension) was thought by Goldstein to occur in patients with an intact speech area. Wernicke's and Broca's areas and the connections between them are preserved in this condition. The connections, however, of this intact speech area to the remaining cortical regions are destroyed by a large lesion in the posterior parietal region. We have seen one patient, followed on this service for several years, who exhibited this syndrome in life and showed at post-mortem the findings I have just cited.

As we have noted, echolalia may occur in the trans-cortical aphasias but is uncommon or at least not prominent in those patients with lesions directly involving the speech areas. Echolalia occurs in many but not all conditions where mutism may occur, e.g., in schizophrenics. It can be seen in certain general disturbances of the brain, such as recovery from coma (it was common when insulin coma was still widely used), or after head injury. It may be seen, as I have noted, in large frontal lesions. It occurs in a group of mental defectives who learn language but make little use of it as a propositional tool.

It should be noted that the properties of the echolalia itself are of little help in assessing the underlying cause. As Stengel brought out so well in his article, most cases of echolalia exhibit the same functional features regardless of their origin.

III. PALILALIC DISTURBANCES

The tendency to palilalia, i.e., to repetition by the patient of words or phrases, is well recognized in certain psychotic patients. An aphasic with markedly limited speech may have a single recurrent utterance, but the palilalic psychotic may demonstrate his tendency in a large variety of phrases.

Palilalia in neurological disease is usually not associated with aphasia. Patients with Parkinsonism may demonstrate this abnormality. Palilalia is a particularly dramatic characteristic of the speech of the general paretic

although it is difficult to detect once the patient has developed a full blown babbling dysarthria. The classical test employing such phrases as 'Methodist Episcopal' or 'hippopotamus' is very effective in eliciting palilalic responses such as 'hippopopototototamus'. Less dramatic palilalia may be seen in other patients with progressive frontal disease. Palilalia, however, need not be confined to syllables as it is ordinarily in paretic speech but may involve whole words or phrases. Any variety of palilalia may occur paroxysmally in seizures involving the supplementary motor area (which lies in the frontal parasagittal region anterior to the motor cortex) as was pointed out in an excellent paper by Alajouanine and his co-workers (1959).

Palilalia, particularly when marked, as in the 'babbling dysarthria' of the general paretic, may lead the observer mistakenly to believe that he is dealing with one of the aphasias marked by fluency and paraphasia, as exemplified by the classic Wernicke's aphasia, in which the patient may also produce a stream of incomprehensible speech. The general paretic will of course show the other stigmata of his disease which considerably simplifies the differential diagnosis.

IV. OTHER CAUSES OF FLUENT INCOMPREHENSIBLE SPEECH

As we have just noted, the marked palilalia of the general paretic may be confused with the classic Wernicke's aphasia. Other conditions may equally present with fluent incomprehensible speech that may be misleading. Thus the pseudobulbar dysarthric patient, much more frequently than is appreciated, shows a *rapid* dysarthria which may be totally incomprehensible. If the patient is forced to slow down (which is often very difficult to achieve) the speech may be recognized as non-aphasic. Such patients normally write correctly.

What was once a more common problem was that of differentiating schizophrenic word-salad from the Wernicke type of aphasia. This problem has become less frequent in recent years for the reason that schizophrenic word-salad is far less common in well-run mental hospitals. Certain features make the differential diagnosis straightforward. In the first place fluent aphasias occur mostly in older patients and are less frequently seen in younger ones and never in children. Schizophrenia by contrast nearly always has its onset below the age of 40, an age at which fluent aphasias

are rare. Furthermore, schizophrenic word-salad is almost never seen in the acutely schizophrenic patient but is most typically seen in the chronic back-ward patient with a long history of behavioral abnormality and hospitalization. The acute onset of fluent incomprehensible speech in older people is therefore almost invariably the onset of fluent aphasia and not of psychosis. Schizophrenic word-salad has certain other features which are helpful in differentiation. It is likely to be filled with Klang-associations and rhymes. It may take a highly regular form, such as adding a fixed nonsense syllable to the end of each word. Secondly, schizophrenic word-salad may be confined to speech while writing is intact, while aphasic patients with fluent speech never have normal writing. The word-salad of the schizophrenic may, in other situations, be present in writing as well as speech or indeed be confined to writing alone. Certain features of word-salad in writing immediately mark it off from aphasic agraphias. One of the major features is that the schizophrenic will tend to fill pages of writing. Such graphorrhea is extremely rare even in aphasics who manifest gross logorrhea. The fluent aphasic often has excellent orthography and his writing may contain many paragraphic errors, but he will almost never run on for pages. In addition, written word-salad will tend to show the same features seen in spoken word-salad, i.e., Klang-associations, rhyming, or the tendency to addition of a fixed syllable to every word (an excellent example of such a patient is given by Bastian, 1898, who apparently did not fully recognize the origin of the disturbance). In addition, the writing may be arranged in carefully worked out bizarre geometrical patterns.

Yet another cause of fluent incomprehensible speech is glossolalia (Kelsey, 1964) or 'the gift of tongues'. Members of certain religious groups at times have episodes of speaking in strange tongues such as are recorded in the New Testament. These people are not in fact likely to represent a diagnostic problem since they do not ordinarily come to the attention of the physician. In any case, the alteration of speech is episodic with normal speech between the periods of glossolalia.

V. GRAMMATICAL DISTURBANCES

Grammatical disturbances in aphasia are most strongly linked to aphasias with anterior lesions, in particular to the classic Broca's aphasia. The

patient, if he is capable of connected speech at all, is typically 'telegraphic', that is to say, he tends to produce speech in which there is a marked tendency to omit the small 'connective' words. Aphasic telegraphic speech is always slow in rate, usually associated with articulation defect and with a tendency to repeat words (Howes, 1964). I have seen one instance of hysterical pseudo-agrammatism in a patient who had been arrested for passing bad checks. While in jail he had developed a disturbance of speech and was transferred to a mental hospital. On examination he spoke in much the same manner that Indians generally do in Western films, e.g., "Me don't know why me here. Me wake up in jail. Me come hospital." What was striking about this speech was that despite the extreme agrammatism, the rate was normal and the words were well articulated except for a marked stutter (which had developed at the same time as the agrammatism). The combination of *rapid* agrammatic speech together with stuttering was unique in my experience. In addition he showed no hemiplegia (as is usually present in most aphasics with telegraphich speech since they have anterior lesions) but did manifest certain other signs strongly suggestive of a hysterical illness. He had a total island of amnesia for the passing of the bad checks and in addition had a Ganser syndrome, one of the classic forms of hysterical pseudodementia. The diagnosis of functional disease received strong support when the patient, a day or two later, showed rapid clearing of all of these signs when he was reassured by his wife that their child would not be taken away by the court in the event of his conviction.

VI. NON-APHASIC MISNAMING

As I have commented earlier Edwin Weinstein and his co-workers (Weinstein, 1958; Weinstein and Kahn, 1955; Weinstein and Keller, 1964), have clarified this syndrome for us. The major diagnostic problem is that of distinguishing this disturbance from a classical amnestic aphasia, a disorder resulting from a localized abnormality in the left posterior parietal region, and often accompanied by neighborhood signs such as a Gerstmann's syndrome or a hemianopia.

Non-aphasic misnaming does not occur in patients who are fully alert, oriented and who readily admit their disability, while anomic aphasia can certainly occur in such circumstances. The patient with non-aphasic mis-

naming may show evidence of gross confusion, drowsiness, memory disturbance, or signs of a widespread dementia; on the other hand he may show only subtle alterations in his state of consciousness or general awareness but is never fully normal in these respects. His naming disturbance has certain characteristics. In some instances he may fail to name even very simple objects and may produce paraphasic errors. The aphasic who manifests such a marked degree of misnaming will show marked word-finding difficulty in his spontaneous speech with a strong tendency to circumlocution. In non-aphasic misnaming, however, there is often a marked discrepancy between the patient's failure to name or his incorrect misnaming and his production of normal speech. One of the striking features of this type of misnaming is that the patient may show a selective difficulty in naming things related to his illness. Thus the patient may name even quite complex objects correctly and yet fail to name things such as 'hospital', 'doctor', 'nurse', 'thermometer'. This selective failure may at times mistakenly lead the physician to assume that the patient is disoriented. The selective misnamings of items related to the illness may have a gallows humor quality. Thus one patient of mine, asked where he was, said, "In a slaughter-house," and asked to indicate the occupation of one of his physicians, said that he was a "grinder." This type of selective misnaming is absent in the true aphasic. Indeed it has often been my impression that many aphasics perform better on subjects related to their illnesses. Non-aphasic misnaming may sometimes make it impossible to detect the presence of a concomitant mild aphasic anomia.

The patient with non-aphasic misnaming frequently shows marked denial of his illness, asserting that he is not ill or that he has come in for some trivial complaint, totally avoiding and frequently actively refusing to admit his major problems.

This clinical picture of non-aphasic misnaming is seen in such conditions as congestive heart failure, uremia, delirium tremens, terminal carcinomas and other diseases with widespread effects on metabolism. Whether the same clinical picture can be reproduced by restricted focal lesions anywhere in the nervous system is not certain. In any case, because of the nature of the illnesses which generally produce this picture, such patients are more likely to be seen by the internist than by the neurologist.[3]

I have seen one patient who exhibited a hysterical anomia. She exhibited no impairments of intellect or consciousness but failed to name very simple objects. Had she been suffering from a true aphasic anomia her spontaneous speech would have been noticeably abnormal; instead her spontaneous speech was perfectly normal without circumlocutions or word-finding difficulties even when she was discussing material which demanded the use of a quite sophisticated vocabulary.

VII. PAROXYSMAL SPEECH DISORDERS

The presence of a clear-cut stable aphasic syndrome assures the examiner that he is dealing with disease affecting directly or indirectly (e.g., via pressure or edema) the speech regions of the brain. Difficulties in speech occurring paroxysmally as part of a seizure disorder may, however, be derived from much wider areas of the brain. Thus seizures in the classic speech areas usually produce arrest of speech (although as Foerster, 1936, pointed out, runs of fluent incomprehensible speech are in fact seen rarely). Penfield (cited by Serafetinides and Falconer, 1963) has obviously described what appears to me to be fluent aphasia following a seizure. Arrest of speech may, however, be seen from sites other than the classical speech regions, e.g., the supplementary motor area (Penfield and Roberts, 1959). Indeed *transient* aphasias may be seen after cortical excisions in this neighborhood as Schwab (1927) pointed out. As Alajouanine and his co-workers (1959) pointed out, the presence of palilalia in the paroxysm is characteristic for seizures in this location. It seems likely that such disturbances occur from either the right or the left side.

Serafetinides and Falconer (1963) have recently asserted that what they refer to as 'paroxysmal dysphasia' (restricting this term to speech arrest and not including fluent paraphasic speech) is overwhelmingly a sign of left-sided disease but that "ictal speech automatisms" (i.e., well-articulated phrases occur during a seizure) come more often from the right side. I suspect that a further distinction may be possible. I would guess that 'automatisms' in which the patient produces paraphasic circumlocutory utterances (like the case of Penfield cited earlier) would result predominantly from left-sided disease, while automatisms in which speech production was normal would result from right-sided lesions.

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[1] It is worth commenting here that we are not concerned with the common difficulties in localization which result from distant effects of a primary disease process, such as occur particularly with space-taking lesions. No neurologist is surprised to see a hemiplegia in the presence of a large cerebral tumor, regardless of its location. because of the effects of displacement of nervous structures, traction on blood vessels, herniations and cerebral edema. Similarly a focal aphasic syndrome may occur in the presence of large space-taking masses and provide only poor information as to the site of the primary disease. What we are concerned with primarily here is the distinction between clinical syndromes which occur with delimited disease and those which require more diffuse involvement.

[2] I am not raising here the problem of the child who never learns to speak at all.

[3] In their earlier papers Weinstein and his colleagues tended to use the term 'paraphasia' to denote non-aphasic misnaming. This usage is somewhat confusing since paraphasic speech certainly occurs in many aphasics with left focal retro-Rolandic lesions. In a more recent paper (Weinstein and Keller, 1964) this terminology is not used; this paper is essentially an excellent summary of the criteria used to distinguish the paraphasias in focal aphasic lesions from those due to disease outside the speech regions.

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CHAPTER VII

THE DEVELOPMENT OF THE BRAIN AND THE EVOLUTION OF LANGUAGE

PREFACE

There exist many theories of the evolution of language, and many were produced in the 19th Century. These theories tried to account for the production of language. They all lead to a paradox. If a mutation occurred which made a man capable of speech, he would have no language to speak nor anyone to understand him. If a mutation made a man capable of comprehension what language would he learn and who would speak it? The possibility of simultaneous mutations in two men in contact seems highly unlikely. More recent theories which stress the origin of language through gesture face exactly the same paradox.

The paper presented here avoids this paradox by showing that language could have evolved through man having learned to 'comprehend' the natural stimuli of the environment. The major step would be the appearance of the ability to associate one specific stimulus to another specific stimulus. Although sound stimuli were probably of special importance this ability probably extended to all modalities.

This paper remains one of my favorites, although the speculations in it may well be wrong, since it is perhaps the only theory in existence which tries to relate the mechanism of some, although not all, aspects of language specifically to the structure of the brain. As I have said the theory may be wrong, and it has been criticized quite harshly. I would welcome some really cogent criticisms since these might well serve either to refine the theory or to replace it by a more sophisticated neurological picture. Unfortunately some of the published critiques have merely been trivial. Thus the statement has been made that the theory would predict that someone born deaf could not acquire language. This is, of course, based on a misunderstanding since the theory assumes that man is distinguished by the capacity to form associations easily between any two non-limbic stimuli. This would obviously include visual-visual associations.

Another criticism of the theory said that the attempt to prove it would involve proving the null hypothesis; since this is impossible, the theory therefore cannot be proven. I found this criticism most curious since it seemed to me to state only the well-known and by now trivial fact that no theory, neither this nor any other, can be proven by any set of facts.

Another criticism, an interesting and important one, attacks the view in the paper that one learns names of things by associating, let us say, the heard name to the seen object. It is argued that in fact names do not represent particular physical objects since one can, for example, immediately understand the word 'knife' applied to an object which has never been seen before. The important point here is to realize that one must make a distinction between the mechanism of the original acquisition of comprehension of names and the later mechanisms. Thus it seems to me likely that at the beginning the learning of names does consist of the association of physical stimuli to names. Later on, however, the brain reorganizes the data according to new hypotheses. By the time one has reached full adult life many names do represent much more complex concepts, and no longer correspond, as they once did, to a simple associative mechanism.

Another objection that has been advanced against the theory in this paper, and indeed in many of the other papers I have written, is that it is associationistic. It is then argued that since, as is well-known, Associationism has long since been adequately refuted, this theory cannot be valid. This argument is based on a confusion of two meanings of the word 'association'. What was refuted was the theory of *Associationism* (note the uppercase letter A), i.e., the belief that all mental processes could be accounted for by frequency, recency, contiguity, etc. The error in this theory was that it neglected the possibility that the nervous system performs major transformations of the data. The refutation of philosophical Associationism should not be confused with the denial of existence of associations, i.e., that in fact the word 'red' may convey certain visual impressions to us. What my theory attempts to do is simply to postulate that certain kinds of associations which are formed more readily in man are essential for language, and it attempts to give an anatomical basis for the ready formation of such associations in man. In any case the need remains of critiques which will lead to replacement or

refinement of this theory, since I continue to suspect that a real understanding of language will not be achieved until we have a reasonable notion of its neurological mechanisms.

THE DEVELOPMENT OF THE BRAIN AND THE EVOLUTION OF LANGUAGE*

1964

ABSTRACT. Certain aspects of the anatomy of the nervous system in man and other primates were reviewed. The distinction was pointed out between the **limbic system** (those structures lying in general in the 'core' of the brain and involved in the motor and sensory aspects of feeding, drinking, elimination, aggression, flight and reproduction) and the **non-limbic** regions of the brain. It was pointed out that anatomically the major connections in subhuman primates from the non-limbic sensory zones (vision, touch, audition) are to the limbic regions and that therefore learning in these animals should consist mostly of associations of non-limbic to limbic stimuli. By contrast the non-human primates lack adequate anatomical structures for the development of associations between non-limbic modalities, and in fact such associations are not formed readily in them.

It was then pointed out that object-naming is in fact acquired by learning an association between two non-limbic stimuli (visual-auditory) and that the development of language in man has in fact been dependent on his acquisition of the ability to form non-limbic cross-modal associations. Reasons were given for the view that this ability depends on the great development of the **posterior parietal region** in man (the **angular gyrus region**).

There was some brief discussion of evidence in favor of this theory and a very brief mention of the possibilities of extension of this kind of theory to other aspects of linguistic behavior. In particular stress was placed on the importance of studying the process by which a child learns a grammatical rule and experimentally devising techniques for teaching these aspects of language in order to learn the mechanisms of acquisition.

Most students of the problem are agreed that there is something special about human language. The major alternative views may be summed up in two simple categories: (1) There is nothing *qualitatively* distinctive about human language; man only possesses *more* language than lower animals. The opposing group of views state: (2) Only man has language. Both approaches leave us with difficulties. If man has more of something than a monkey then just what particular function of the monkey's is it that is being multiplied in man? If only man has language then what is the essence of this new addition to the armamentarium of the activities of animals?

The first group of theorists are likely not to worry about the details of what it is in the animal that is multiplied in man. They tend to express their

ideas as *Gedanken* experiments: Tell me what you want the animal to do and I can devise a training procedure to do it. The second group tend to phrase the differences in terms sometimes as ethereal as those of the first group are uncompromisingly untheoretical and practical.

Regardless of their particular position of the adherents of each of these views have shared in common either an inability or a lack of interest in specifying what change in the design of the human brain permits of these quantitative or qualitative distinctions. This paper represents an attempt to bridge this gap in some small way. The linguist, even if he accepts the views presented here, may well find them disappointingly meager. The first day of this conference has been devoted to a series of papers representing much of the forefront of pure linguistic theory, and the overwhelming stress has been placed on aspects of syntax. The linguist would certainly wish to know in what way the human brain has become adapted to the production of syntactic speech. Instead he will have to be satisfied – if indeed he is satisfied at all – by a theory of object-naming. Let me therefore hasten to state that I make no pretensions for what I say except insofar as it applies to object-naming. On the other hand an interest in object-naming is not ill-placed since much of the practical use of language rests on the basis of this rather unglamorous activity. Even object-naming in itself has its more interesting sides. Thus, workers in childhood development have tended to neglect the acquisition of names since this has been regarded as the simple enlargement of the lexicon, and have concentrated on the evolution of grammar. Yet the problem of the lexicon goes beyond the mere filling of the dictionary randomly. Thus, the naming of colors, which to the adult appears like a remarkably simple task, is often acquired late, long after the child can name remarkably complicated objects. The ramifications of this extend still further into language performance. Thus, there are reasons for regarding it as not unlikely that the time of acquisition of color-naming and of reading should be highly correlated.

There is perhaps one more reason to stress object-naming. While the mechanism suggested here for the naming of things throws no direct light on the acquisition of grammar, it does throw into relief some different ways of looking at the acquisition of grammar which may well be experimentally useful.

It is perhaps appropriate to put in one final comment on this type of

theorizing. The linguist may wonder whether such a theory, even if correct, is any concern of his. Let me suggest some reasons which justify interest by linguists in this type of theory. In the first place it is reasonable that anything which throws some light on the nature of language should be of interest to the linguist. A second reason is that the linguist has the ability to provide help to the student of the nervous system. The theory of object-naming to be propounded here has certain implications which might conceivably be more effectively tested in a non-Indo-European language.[1]

There is a third reason which is perhaps the most cogent one for the linguist to take some interest in these matters. I gather from various sources, for example, from the proceedings of this conference a year ago, that the status of linguistic theories continues to be a difficult problem. In a sense the difficulty reduces to that of deciding whether the best theory is simply the most economical set of axioms from which the language behavior can be deduced, i.e., in essence a condensed description. If this is true then theoretical linguistics might appear to be only a more efficient form of description. I would wish, cautiously, to make the suggestion, that perhaps a further touchstone may be added: to what extent does the theory tie in with other, non-linguistic information, for example, the anatomical aspects of language? In the end such bridges link a theory to the broader body of scientific knowledge. I would personally not see much virtue in the views of these theoreticians who feel that language and its disturbances must be viewed *separately* on the linguistic and biological levels. Let us now return to the main topic of this paper.

In order to specify what the differences are between man and subhuman primates let me first discuss some of the features of learning in these non-human forms. I hope I may be forgiven for some preliminary remarks about some elementary anatomical and physiological aspects which may not be familiar to all of you. If the discussion *appears* very remote from our topic please rest assured that it is all really germane.

Lying along the inner surface of the temporal lobes of the brain are a group of structures of extremely complex anatomy. For the sake of simplicity I will loosely use the term 'limbic system' to refer to these and to their major connections in the **hypothalamus** and other structures lower in the brain. Included in the anatomical organization of this region are those parts of the brain which when stimulated give rise to sensations of

taste or smell, and to certain emotions such as fear and their corresponding motor activities. For simplicity we may divide those aspects of the limbic system of interest to us into two groups: (1) **Limbic motor responses** – these are highly organized, essentially inborn motor sequences which mediate the motor responses involved in fear, rage, and sexual activities. (2) **Limbic sensory responses** – these are the subjective feelings of smell, taste, hunger and thirst and the corresponding feelings of satiation, sexual sensations, anger, and fear. It is not at all inaccurate to say that the limbic system mediates both the inborn motor sequences involved in those elementary activities initimately related to the survival of the organism or the species and the subjective experiences related to these activities.

Let us now consider by contrast the remaining 'non-limbic' portions of the surface of the brain which, in fact, make up the bulk of the exposed surface of the hemispheres in the primates, including man. In particular we will concentrate on the sensory portions of this non-limbic brain. There are three sensory regions which are to be specified here: the **visual cortex**, the **auditory cortex**, and the **somesthetic cortex** (which eventually receives the sensory information from the skin, muscles, bone, joints, and tendons – the term 'touch' can loosely be used to designate this group of sensations). These three regions may be said to mediate the 'non-limbic' modalities of sensation.

The zones we have so far considered, that is the 'limbic' regions on the medial surface of the temporal lobe, and the primary centers for the 'non-limbic' sensations of vision, audition and somesthesis, constitute only a small fraction of the surface of the hemisphere. These four regions, together with the classic **motor cortex**, are all, in the terminology of the great German neuroanatomist Flechsig, 'primordial zones', that is, regions which mature early in the development of the individual. The most commonly used criterion for such maturation is the degree of myelination of the nerve fibers in these regions. These primordial zones also differ quite markedly from each other in cellular architecture.

If we contemplate the brain of a subprimate mammal, such as a rabbit or a cat, we find that these primordial zones make up the majority of the cerebral mantle. As we move up the phylogenetic scale we find the striking fact that these zones become increasingly separated by new areas of cortex. The cortex separating the primordial zones occupies a greater percentage of the surface in the primates and achieves its greatest extent

in man where it clearly occupies most of the surface of the hemispheres. It is indeed the development of this **association cortex** which is responsible for the higher functions of the nervous system. The association cortex matures later in the life of the individual. Flechsig called these regions 'intermediary zones' to indicate their later myelination. The different parts of the association cortex are more like each other in structure than are the different primordial zones.

Let us now turn to the problem of the connections between these different regions of the cerebral mantle. Flechsig (1901) stated an important principle for the brain of man which has in recent years been reemphasized for the brain of the subhuman primate by Bailey and von

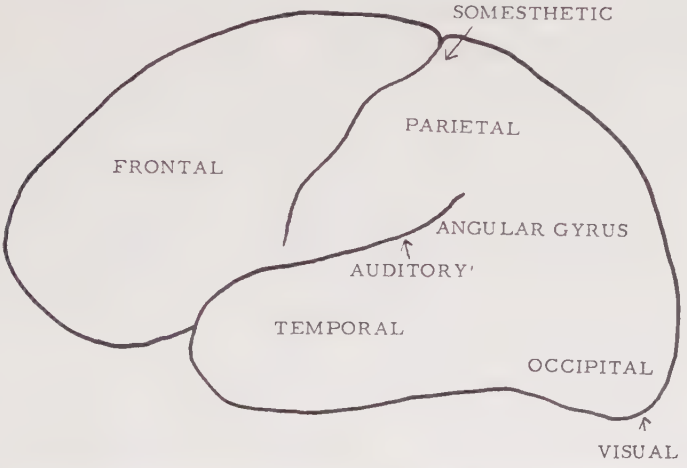

Fig. 1. View of lateral (outer) surface of human left cerebral hemisphere. The frontal, parietal, temporal, and occipital lobes are indicated. In addition the arrows point to the primary somesthetic, auditory, and visual regions of the cortex. The region of the angular gyrus is also shown. Fuller explanation is given in the text.

Bonin (1951). The principle asserts that in man the primordial zones, i.e., the regions of early maturation, do not have any significant number of direct connections between them. Thus there are no significant direct interconnections between the limbic regions, the motor cortex, the auditory, somesthetic, and visual cortexes.[2]

Each primordial zone has a significant number of connections only to

the immediately adjacent association cortex. The association cortex itself may have long connections to other regions. Let us now consider the connections of the non-limbic sensory modalities. In particular let us consider the connections of the visual cortex which are the best known and may serve as a paradigm for the others.

The primary visual cortex makes connections to the immediately adjacent visual association cortex. The visual association cortex has three important sets of connections, the first two of which I mention only in passing, and the third of which is immediately germane to our argument: (1) Connections to the visual association cortex of the opposite side via the **corpus callosum.** (2) Connections to the association cortex anterior to the classic motor cortex. (3) The largest connection and the one of most concern to us runs from the visual association cortex to the outer and inferior surfaces of the temporal lobe.

Why should the largest connection of the visual cortex eventually reach this portion of the temporal lobe? If we consider in turn this region of the temporal lobe we find that it makes connections with the limbic structures on the inner surface of the temporal lobe. In fact the limbic structures themselves constitute one of the primordial zones and therefore do not receive long connections directly from any other region of the cerebral cortex but receive connections only from the nearby association cortex. The lateral and inferior temporal lobe is thus the association cortex for the limbic structures.

Let us now sum up briefly the point to which these considerations have led us. The visual cortex has its main connections to the limbic system. These are not direct connections but go by way of the association cortex adjacent to the visual cortex and the association cortex on the lateral and basal surfaces of the temporal lobe. We are now prepared – at last – to consider the functional significance of these anatomical connections.

In order to approach this problem let us ask ourselves how a monkey would respond with a rage response to the sight of an object (such as a net) which he has learned to dislike. The impulse must travel over the pathway I have just specified from the visual cortex to the limbic structures and there arouse the inborn rage response. Suppose this pathway is broken at some point? The monkey will no longer respond with rage to this visual stimulus. We will have produced a visual-limbic disconnection.

Consider now an example which is far more important for our purpose.

How do we teach a monkey presented visually with, let us say, a circle and a cross to press the lever under the circle rather than that under the cross? We do this by what the psychologists call *reinforcing* one or another response. We may reinforce positively, i.e., reward the response we wish the animal to make, or we may reinforce negatively, i.e., punish the response to be rejected. I do not wish to go into the complexities of the question of reinforcement. For the sake of simplicity we may say that the primary reinforcers are limbic sensations, i.e., the rewards are such things as the taste of food, the satisfaction of hunger or thirst; the punishments are such things as the arousal of fear.

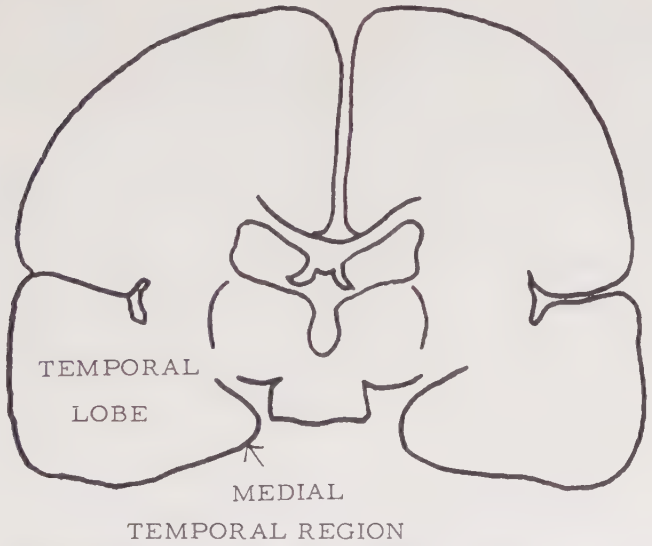

Fig. 2. Cross-section in the vertical plane through the human brain. The temporal lobe is indicated. The medial temporal region (whose importance is discussed in the text) is also shown.

In effect then the visual learning paradigm in the monkey may be reduced to a simple formula: the animal learns to form associations of a visual stimulus to a 'limbic' stimulus. In more general terms learning in the monkey consists in forming associations between a 'non-limbic' stimulus and a 'limbic' stimulus. This is true for vision, audition, and somesthesis. From the point of view of evolution this is reasonable: a

stimulus in a non-limbic sensory modality is learned readily only to the extent that it arouses a sensation important to the survival of the individual animal or the species.

In order to look a bit more deeply at learning in the monkey let us now turn to a somewhat different test situation, the one reported by Ettlinger (1960). Here the monkey is taught to choose one of two patterns presented visually, let us say, a circle rather than a cross, by the technique already mentioned of rewarding the choice of one item. After the monkey has learned this to a high degree so that he chooses the rewarded circle on nearly all occasions, another experiment is performed. The monkey is now permitted to palpate two solid figures, a circle and a cross respectively, which are concealed from vision. The animal is again rewarded for choosing the circle rather than the cross. Curiously enough he shows no evidence of carry-over from the identical task done visually. The tactile task is treated by the monkey as a totally new one.

How are we to interpret this experiment and others which have essentially the same result? Ettlinger and others have argued that cross-modal transfers do not tend to occur in animals. But our previous discussion has already stressed that *all* learning in the monkey is cross-modal since the process of learning depends on the formation of associations between *non-limbic* and limbic sensations which is certainly a cross-modal task. The essence indeed of these experiments is that the animal has failed to form an association between two non-limbic modalities.[3]

The same task is, of course, remarkably easy in man who makes the transfer immediately in at least this particular experiment. What is the reason for the ease with which humans do this task which is so difficult for the monkey? Some authors say that man does it by means of verbal mediation. In other words, when one learns to choose a circle over a cross one learns to choose the 'circle'. When the task is done we again choose the object which we have named 'circle'. The monkey's failure at these particular cross-modal tasks is thus attributed to his failure to use language.

A moment's reflection, however, shows that this argument which appears so plausible at first glance, has in fact been stated backwards. In order to call a seen object a circle we must have learned to associate the visual stimulus with the auditory stimulus 'circle'. In other words, to say that the ability to perform cross-modal associations depends on the ability

to name misses the real point which is, as it were, the reverse: *The ability to name depends on the ability to form non-limbic cross-modal associations*, particularly *visual-auditory and tactile-auditory*.[4] One may go further: *The ability to develop language in man probably depends on his ability to form cross-modal associations* between two non-limbic modalities. One might raise the objection, however, that a deaf man might learn language without using cross-modal associations at all. Could he not learn to read by being shown the picture of the object which corresponded to each new written word? Indeed he probably could. In order, however, for him to be able to name (in writing) things with which he palpated he would at least have to be able to form tactile-visual associations. There is, however, a further consideration. Even a visual-visual association is a link between two non-limbic stimuli, and it is questionable that even this would readily be acquired by the monkey.

We have asserted then the thesis that man develops language because he can form associations between two non-limbic stimuli. Couldn't limbic stimuli be useful as language? The answer of course is no: a monkey would have a great deal of difficulty arousing in another animal a smell or taste, or a feeling of hunger or thirst. We communicate by producing non-limbic stimuli – auditory, visual, or tactile.

What is the basis for the difference in the ability of man and the monkey to form pure non-limbic associations? Let us return to our anatomical considerations. As we have pointed out there are three main connections from the visual association areas of a monkey – one to the symmetrical region of the opposite side, one to the motor association area, and the largest to the lateral and basal temporal lobe which is the association cortex for the limbic system. Thus none of the *main* connections are designed to mediate visual-auditory associations. There are, however, definite connections between some of the visual and auditory association areas, as shown for example in the experiments of Sugar *et al.* (1948). These experimenters found clear evidence of fibers running from the auditory association cortex to the visual association cortex but none in the reverse direction; in other words they found no evidence for fibers which could mediate the arousal of auditory memories from a visual stimulus. Even the auditory to visual fibers they found constitute a group far smaller than the fiber systems mediating visual-limbic associations. The monkey brain thus probably contains visual, auditory, and somesthetic

regions which, operating on the whole independently, feed into the limbic system stimuli which are used only to the extent that they more or less immediately affect survival.[5]

Even if a small number of fibers exist which have not been discovered between the visual and auditory regions, this would not affect the argument. The more information that must be carried between two cortical regions, the more fibers necessary to carry them. The more associations formed, the more cells are needed to store the memories. The monkey does not appear to have a sufficient basis anatomically to form extensive non-limbic intermodal associations. That a limited number of such associations could be performed perhaps with extensive training is probably correct, but there is probably an inherent limit to the speed of formation or the variety of such associations. One might say that the monkey lacks adequate 'memory space' and 'channel capacity' for this type of association.

It might be worth digressing for a moment to consider the great strain put on the facilities of a brain by a demand for non-limbic intermodal associations. Consider the situation that visual, auditory, and somesthetic association areas discharge independently into the limbic system via association cortex. In order to add visual-auditory and visual-tactile associations to the visual-limbic associations one could probably have to add at least twice as many fibers and cells are now used for the visual-limbic connections alone. The probable minimum net effect of such an extensive addition of non-limbic intermodal fibers is therefore to triple the array of cells and fibers needed in the visual association system. It is no surprise that this uneconomical situation does not appear even in the subhuman primates.

What then of the human brain? What correlates with the newly acquired or at least greatly increased ability to form non-limbic intermodal associations and thus to develop language?

It is obvious that there has been marked development of the human brain in comparison to that of even the highest apes (Connolly, 1950). Thus the gorilla has the largest brain of any of the anthropoids, but it never even reaches half the weight of an average adult human brain. In addition the **cerebellum** makes up a higher percentage of the brain in the anthropoid apes so that the cerebral hemispheres of man (which constitute the part which is of concern to us) are relatively even larger than the

discrepancy in whole-brain weights would suggest. Furthermore, a fully adult gorilla is a considerably larger animal than a human. An orangutan which when fully grown may weigh as much as a small human adult has a brain which is about one quarter as heavy as the human brain at maturity.

The human brain at birth is perhaps 40% of its adult size while that of the ape's is nearly 70% of full-grown size. This suggests that much of the human brain is evolutionarily late since it matures so late in the life of an individual. This supposition is amply borne out by closer inspection. The great increase in the size of man's brain is primarily in the association areas, rather than in the primary receptive areas. This growth is reflected in the **frontal pole**, the **temporal lobe**, and in the **inferior parietal region.** The greatest relative growth of the human brain compared to that of the subhuman primates is in the inferior parietal region, and it behooves us to look at this zone more carefully.

The impressive development of the inferior posterior parietal region (the region of the **angular gyrus**) is so great that some authors (e.g., Goldstein, 1927) even assert that this region is almost unique to man. Even those who deny this, such as Bonin and Bailey (1961), still stress that it is this region of the brain which has expanded most markedly in man compared to any of the apes. Its advanced state is testified to by the fact that it is one of the last regions to myelinate and was called by Flechsig one of the 'terminal zones' in contrast to the earliest myelinating 'primordial-zones' and the 'intermediary zones' which myelinate at an intermediate period. According to Dr. Paul Yakovlev this region does not mature in its cellular structure until three or four years or even much later in childhood.

This region is ideally located for the purposes of our theory. It is placed between the association cortexes of the three non-limbic modalities: vision, audition, and somesthesis. It is thus admirably suited to play the role of acting as the way-station by which associations may be formed between these non-limbic modalities. This area may well be termed 'the association cortex' of the association cortexes. By providing the basis for the formation of non-limbic associations, it provides the anatomical basis for language – or at least for object-naming. Other authors have stressed the importance of the posterior parietal region for language,[6] but I hope that I have been able to give a physiological basis to this localization.

There are many problems associated with the discussion I have just presented. Indeed, rather than use the word 'problems', I would perhaps have been more correct in saying that it raises many possibilities for further investigation. Much of the data now available does support the view advanced here. I will cite only a few examples of such data.

Note to the reader. The following section contains rather technical material which can be omitted without violence to the main trend of the argument.

Very large lesions of the angular gyrus region produce an aphasic picture in which the patient speaks volubly but communicates little information. The speech is in particular remarkably poor in the names of objects. The reason for this clinical picture is readily deducible. The connections from the rest of the brain to the auditory association area are cut off. On the other hand since the auditory association area and the motor speech region and their connections are intact, the patient can still produce the automatisms of language freely – these are those aspects of language not demanding intermodal transfer for their use at a given instant. The comprehension of language is seriously impaired since the arousal of associations in other modalities by the auditory speech patterns is also cut off by the lesion. These patients, despite severe comprehension defect, may show perfect repetition because they have preserved the connections between the auditory association cortex and the motor speech region.

Even more revealing as to the importance of intermodal associations are certain more limited lesions. Thus in certain partial lesions of the angular gyrus (first described by Dejerine, 1891) there may be an isolated loss of the ability to comprehend written language and to write (the so-called 'pure alexia with agraphia'), while all other aspects of language are intact. The functions of the angular gyrus in auditory-visual associations may be quite dramatically brought out by considering an even more restricted lesion, that of the so-called 'pure word-blindness without agraphia' (Dejerine, 1892; Geschwind, 1962). Here the left primary visual cortex is destroyed. The patient can thus see only with his right visual cortex. In addition, however, there is usually a destruction of the posterior end of the corpus callosum. This acts to cut off the right visual region from the left angular gyrus region, and thus the patient

cannot understand written language. All other aspects of language are intact.

The disturbances in these patients can be summarized as follows: they lose the ability to comprehend written language, to read music, and to name colors (but they can still match colors correctly and show other evidences of correct color perception by *non-verbal* means – see discussion in Geschwind, 1965). They tend to preserve the ability to name objects and to read numbers. How does one make sense of this constellation of impaired and preserved visual tasks? Reading is learned nearly always after speaking. While one learns in speaking to name objects with which one has had both tactile and visual experience, learning to read is almost exclusively dependent on pure visual-auditory connections (particularly after the earliest stages). When these pure connections are cut off the patient can no longer read. He can still copy the words he cannot read (which effectively rules out almost any perceptual theroy of this disturbance) because visuo-motor connections in the right hemisphere are still intact. He can name objects because these arouse tactile associations further forward in the right hemisphere and a connection can be made to the speech regions across an intact part of the corpus callosum lying forward of the damaged part.

Color-naming and music-reading are lost with word-reading because they are very pure visuo-auditory tasks – we can identify an apple by its form or texture, but we cannot identify a color by these. Finally, number-reading is preserved because numbers are usually associated with a powerful somesthetic reinforcement (counting on the figures) through a considerable part of childhood, a form of reinforcement not available for words.

The thesis presented here is that the development of object-naming depends on the presence of anatomical structures in man which are absent or poorly developed in the monkey. The assertion that object-naming depends on visual-auditory transfers is of course an old one in linguistics. Wittgenstein (1953) opens his *Philosophical Investigations* with a quotation from St. Augustine's *Confessions*: "When they (my elders) named some object and accordingly moved towards something, I saw this and grasped that the thing was called by the sound they uttered when they meant to point it out...."

Can we expand the theory presented here to more than object-naming

and can we somehow use the same approach to help us with grammar? We may appropriately quote Wittgenstein's comment on the above passage, "Augustine does not speak of there being any difference between kinds of word. If you describe the learning of language in this way you are, I believe, thinking primarily of nouns like 'table', 'chair', 'bread', and of people's names, and only secondarily of the names of certain actions and properties; and (*sc.* you are thinking) of the remaining kinds of word as something that will take care of itself." But, of course, Wittgenstein implies, and we must agree, that the other words will not take care of themselves. How would you teach a child the use of the word 'if' by the method described by Augustine?

I do not have a solution to offer at this moment. It would be difficult to conceive of grammar as depending on intermodal transfers in the straightforward manner that object-naming does. Grammar appears at first glance to be something that grows up within the auditory association system itself, to depend on intramodal, auditory-auditory associations (which can then be transferred *en bloc* to written language by use of the cross-modal association system we have discussed).

This answer is, needless to say, hardly satisfactory, but our earlier discussion may give a clue as to some useful future approaches. We began this paper by stressing the exact method by which a monkey learns to choose a circle over a cross. Similarly, we have discussed the exact mechanism of object-naming in man. We can answer questions as to how we would teach certain things to a monkey and how we could teach a child the name of an object. Let us consider the possible applications of the method to grammar. We have some important facts, gleaned in important studies in recent years, on the child's acquisition of grammar. These facts, however, are still concerned mostly with the age of acquisition of certain grammatical features, their order of acquisition, and the learning of distinctions between features that might at first be accepted as equivalent. To some extent we can specify what rule the child follows at each age. I would suggest, as a supplement and by no means a substitute to these very important studies, that some attention be paid to the actual act of learning by the child and that some experiments be devised to study the elementary situations in which children learn to use certain words. At its origin grammar may well be acquired by mechanisms as naive as those of object-naming. Once these elementary associative steps

are passed the acquisition of the more complex structures presents less difficulty. It is the very first steps which are the vital ones. Once speech is developed, it can develop an autonomy from the outside world that is all too familiar. If our studies on learning by monkeys and object-naming in man are to be of use, they should teach us to study not what grammatical rule is acquired by the child at a given age but *how* he has acquired this rule and how we could teach it to him.

NOTES

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[1] For example, the theory deals with the problem of color-naming as a corollary. The theory predicts that color-naming, if learned in a certain way, should be affected by certain lesions of the nervous system. In a hypothetical language in which there were no color-names, but in which the color of an object was stated as the color of some other well-known object, the theory would predict that this lesion should not have the same effect. A linguist is more likely to avoid the naive error that color-naming is done in the same way in all languages.

[2] This principle holds to its full extent in man, the monkey, and chimpanzee, and probably in the other primates, at least the higher ones. It does not hold completely in sub-primate forms; for example, it is clearly not true of the cat's brain. It is therefore important to distinguish the species involved and not to generalize too readily to the human brain from that of lower forms, particularly when these lower forms are sub-primates.

[3] An objection might be raised that Ettlinger's experiment actually relates to cross-modal *transfer*, not cross-modal *association*. This problem is too complex to discuss fully at this point. Transfer is probably the simpler of the two, and therefore the failure of transfer probably also implies difficulty in association. In any case other evidence, e.g., from conditioning experiments, also points to the difficulty of nonlimbic intermodal *associations* in subhuman forms.

[4] It is true that in adult man some visual-tactile associations may be done indirectly by the linking up of two separate cross-modal associations (visual-auditory and tactile-auditory). Each of these components is, however, itself an elementary non-limbic cross-modal association.

[5] There is a possible exception in the 'curiosity' drive which has been studied by such workers as Butler (1953) and which does not appear to be quite so 'primitive' as the other limbic reinforcers. Even this drive is very likely to depend on the limbic system.

[6] See for example the discussion in Critchley (1953).

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CHAPTER VIII

DISCONNEXION SYNDROMES IN ANIMALS AND MAN

PREFACE

As I have pointed out earlier, when I met Oliver Zangwill in 1961 at a meeting on dyslexia in Baltimore, he listened patiently to the exposition of my ideas on the significance of the cortico-cortical connections for the higher functions. A short time later, while on a trip to Boston, he suggested to me that I should prepare an extended account of these ideas. This paper would never have been written without Professor Zangwill's urging, and I am grateful to him for having brought me to a more careful review of the older literature and a more precise statement of my own ideas. Although Russell Brain, who was then the editor of *Brain*, had some misgivings about the section on philosophical implications he agreed to take the manuscript unchanged.

In the years since this paper was written I have revised my ideas on some issues and expanded considerably my thoughts on others. I am now preparing a complete revision for publication in book form.

DISCONNEXION SYNDROMES IN ANIMALS AND MAN*

1965

ABSTRACT. A complete summary of all the material presented would be much too extensive and, indeed, much too repetitious. I will therefore try to outline here the major points presented in this chapter. I have attempted to show that many disturbances of the higher functions of the nervous system, such as the aphasias, apraxias, andagnosias may be most fruitfully studied as disturbances produced by anatomical disconnexion of primary receptive and motor areas from one another. For a detailed discussion the reader is referred to the appropriate sections of the paper.

In the lower mammals connexions between regions of the cortex may arise directly from the primary receptive or motor areas. As one moves up the phylogenetic scale, these connexions come to be made between newly developed regions of cortex interspersed between the older zones. These regions are called 'association cortex'. As Flechsig pointed out for the human brain, all intercortical long connexions (whether in or between hemispheres) are made by way of these association areas and not between the primary motor or receptive areas. It follows from this that lesions of association cortex, if extensive enough, act to disconnect primary receptive or motor areas from other regions of the cortex in the same or in the opposite hemisphere.

The connexions of the visual association regions were discussed in some detail, and it was pointed out that the major outflow of these regions is to the lateral and basal neocortex of the temporal lobe which in turn connects to limbic structures. Lesions of the lateral and basal temporal lobe therefore tend to disconnect the visual region from the limbic system. This leads to a failure of visual stimulation to activate limbic responses, such as fight, flight, and sexual approach. It also leads to difficulties in visual learning. These can be thought of as resulting from the failure of the animal to form visual-limbic associations (such as learning that a visual stimulus equals the food reward given for correct choice) because of the lack of appropriate connexions. They can also be regarded as disturbances in visual recent memory resulting from a disconnexion between the visual region and the hippocampal region. The discussion was then applied to the tactile and auditory systems. Learning difficulties in primates involving these systems also were thought to result from disconnexions from the limbic system. 'Agnosia' in the sense of failure to respond to stimuli within a single modality appropriately in the face of intact perception in that modality is regarded as being a part of the syndrome of disconnexion of primary sensory modalities from the limbic system. Since callosal fibres arise from association cortex, failures of interhemispheric transfer may result from lesions of association cortex. The problem of whether disconnexions of single modalities from the limbic system in man occur was briefly discussed.

While connexions between primary receptive regions and limbic structures are powerful in subhuman forms, intermodal connexions between vision, audition, and somesthesis are probably weak in these animals, a view for which evidence is available both on the basis of experimental behavioural investigations (e.g. studies on intermodal transfer of learning and on higher-order conditioning) and on the basis of anatomical evidence. In man the situation changes with the development of the association areas

of the human inferior parietal lobule, situated at the junction of the older association areas attached to the visual, somesthetic, and auditory regions. It is speculated that this new 'association area of association areas' now frees man from the dominant pattern of sensory-limbic associations and permits cross-modal associations involving non-limbic modalities. It is particularly the visual-auditory and tactile-auditory associations which constitute the basis of the development of speech in most humans. In man the speech area (which constitutes the auditory association cortex, particularly that part of it on the convexity of the temporal lobe, also Broca's area and the connexions between these regions) becomes a structure of major importance in the analysis of all the higher functions.

Pure word-blindness without agraphia was then discussed as an excellent, classical example of disconnexion from the speech area; this syndrome results from a combination of lesions, the usual one being destruction of the left visual cortex and of the splenium of the corpus callosum. The association with this syndrome of colour-naming difficulties and inability to read music is noted, along with the relatively strong preservation of the reading of numbers and the naming of objects. Reasons are advanced for these discrepancies. The problem of childhood dyslexia and its associated disturbances and its possible relation to the acquired dyslexia of adults was briefly presented.

Other disorders with similar pathogenesis (isolation of a particular sensory modality from the speech area), i.e. pure word-deafness and tactile aphasia were then briefly discussed.

The problem of the 'agnosias' was then presented. Evidence was presented against the idea that there exist disturbances of 'recognition' regarded as a unitary faculty. It was argued that most of the 'agnosias' are in fact modality-specific naming defects resulting from isolation of the primary sensory cortex from the speech area and associated with marked confabulatory response. A critique was presented of the classical 'aphasic-agnosic' distinction. There was presented some further discussion on the determinants of confabulatory response. The problem of right parietal syndromes was presented in the light of the preceding discussion of the 'agnosias'.

'Apraxic' disturbances were analysed in detail and were regarded as resulting from disconnexions of the posterior speech area from association areas which lie anterior to the primary motor cortex, and from disconnexions of visual association areas from these 'motor association' areas. The problem of left-sided predominance was discussed. In particular apraxic disturbances resulting from callosal lesions, from lesions of 'motor association' cortex and from damage deep to the supramarginal gyrus were discussed. The apraxia of the left side ('sympathetic dyspraxia') of aphasic right hemiplegics was discussed as well as facial apraxia. The sparing of certain types of movement in the apraxias was discussed, particularly whole body movements and was related to the probable preservation of Türck's bundle (whose connexions were presented in some detail) running from the posterior temporal region to the pontine nuclei and then via synapses to the cerebellar vermis.

Finally syndromes resulting from disconnexions within the speech area (conduction aphasia) and the pattern resulting from the isolation of the speech area were presented.

Some classical objections to the disconnexion approach were presented, in particular the results of Akelaitis and reasons for his negative results were discussed. This section closed by pointing out that this type of theory suggests many experiments and anatomical investigations. The dangers of *ad hoc* postulation of connexions were mentioned.

In a short section attention was called to some philosophical implications of these

findings, particularly for the notions of 'regarding the patient as a whole man', the unity of consciousness, the uses of introspection and the relations between language and one's view of the world.

INTRODUCTION

The early successes of the views of Broca and Wernicke led the classical neurologists to a mode of analysis of the disturbances of the higher neurological functions subsequently to be labelled with the derisive term 'diagram-making'. Starting from the picture of the brain as a collection of sets of more or less specialized groups of cells connected by relatively discrete fibre pathways, these classical neurologists deduced a series of symptom complexes. On the basis of this model clinical syndromes could be divided into those resulting from lesions of grey matter and those which resulted from lesions of the white matter interconnecting specialized regions. Thus, cortical syndromes were distinguished from 'conduction' syndromes. Basically it was this mode of analysis which dominated the literature until the First World War.

An interest in the connexions between different parts of the speech region and between the speech region and the remainder of the brain dated back to almost the earliest of the classical writings. Wernicke (1874) had already predicted the existence of a particular aphasic syndrome resulting from disconnexion of the sensory speech zone from the motor speech area by a single lesion in the left hemisphere. Subsequent developments showed him correct in principle although probably at least partially incorrect in his assumption as to the location of the pathway between these regions. These earliest studies concentrated on lesions of white matter separating regions within a single hemisphere. Dejerine (1892) in describing the pathology of pure alexia without agraphia probably was the first to show definite clinical symptomatology as the result of a lesion of the corpus callosum. Hugo Liepmann carried the analysis of syndromes resulting from the disconnexion of specialized regions of grey matter to its most important development. He published the first post-mortem of a case of pure word-deafness from a unilateral lesion (Liepmann, 1898; Liepmann and Storch, 1902) consistent with if not fully establishing the hypothesis that this syndrome resulted from isolation of the speech area from auditory inputs into both the left and right hemispheres. He described the famous case of the Regierungsrat (Liepmann, 1900) in which he

carefully analysed this patient's behaviour and explained it on the basis of a series of disconnexions. In this paper he predicted the sites of the lesions. The post-mortem findings (Liepmann, 1906) amply confirmed these published ante-mortem predictions. One year later he published with Maas the famous case, Ochs (Liepmann and Maas, 1907), which showed the effects of callosal disconnexion on motor function.

In the immediately following years Liepmann's results were repeatedly confirmed by such workers as Kurt Goldstein (1908), Bonhoeffer (1914) and a host of other authors. As Liepmann (1914) himself pointed out, those who were apparently his severest critics, such as von Monakow, had indeed fundamentally accepted his point of view; in fact, no really important criticism was ever directed against his analysis. Goldstein's (1927) monograph on cortical localization showed his continuing acceptance of much of Liepmann's approach.[1]

That Liepmann's analysis of apraxia from the point of view of disconnexions continued to be part of the accepted doctrine of German neurology is evidenced by the article of Lange (1936) in the Bumke-Foerster Handbuch. By contrast, English-speaking neurology showed relatively little interest in this approach. Wilson (1908) summarized Liepmann's classical papers on apraxia accurately, but there was no rush of papers intimately describing cases similar to Liepmann's; Head's (1926) account of apraxia is cursory and, at least in part, incorrect.

On the whole the period between the wars seems to have led to a loss of interest in analyses in terms of disconnexion. The criticisms of the holistically oriented neurologists, Head, Marie, von Monakow, and Goldstein probably contributed heavily to this decline of interest. The growth of holistic psychology under the Gestalt school and Karl Lashley and the rapid development of holistic schools of psychiatry probably all played a role, perhaps more by their effects on the general atmosphere of thought than by their specific critiques of the classical school. The disappearance from the active scene of Dejerine and Liepmann removed two of the greatest contributors and defenders of the older school. When the papers by Akelaitis and his co-workers (Akelaitis, 1941a, 1941b, 1941c, 1942a, 1942b, 1943, 1944, 1945; Akelaitis *et al.*, 1942; Smith and Akelaitis, 1942) on the negative results of callosal section in epileptics appeared, most neurologists, at least in England and the United States, were prepared to reject for good the classical teaching on the importance of commissural and

intra-hemispheric association pathways, a doctrine which had, after all, been losing ground for the previous twenty years. Only occasional papers such as those by Trescher and Ford (1937) and a later one by Maspes (1948) reasserted the importance of the corpus callosum but these were generally neglected.

The tide of interest in the callosum began to turn when Myers and Sperry (1953) showed that the callosally sectioned animal did show dramatic changes in behaviour if appropriately examined. Their work led my colleagues and myself in 1961 to re-examine the older clinical literature and to reassess our patients with disturbances of the higher functions. We were gratified to find that within a few weeks we were able to find two patients whose symptomatology could be attributed to involvement of the corpus callosum. One of these patients showed the syndrome of pure alexia without agraphia (Geschwind, 1962; Howes, 1962). The second showed a much more extensive syndrome which my co-worker, Mrs. Edith Kaplan, and I attributed to an infarction of the corpus callosum. In late 1961 we presented our findings on this patient at the annual research meeting of the Veterans Administration and at a meeting of the Boston Society for Neurology and Psychiatry (Geschwind and Kaplan, 1962a). A more complete study of this patient was published later (Geschwind and Kaplan, 1962b) and we were able to point out in a last-minute footnote that the post-mortem findings had confirmed our ante-mortem prediction of a callosal infarction sparing the splenium. These cases have stimulated us to look further into our clinical material and to delve still further into the literature.

In the pages which follow I hope to give an account of the implications of thinking in terms of disconnexions for both clinical practice and research. The synthesis presented here was developed piecemeal out of study of the literature and clinical observation. I will not, however, present it in the order of its development but rather will try to organize the facts and theories along simple anatomical lines. There is, I believe, a unity in the theory which justifies this approach, and I hope that it will significantly contribute to clarification of the presentation. There are many facts recorded in the following pages; there is also much speculation which is, however, nearly all subject to the checks of future experiment and clinical observation.

While the material of this paper is fundamentally organized phylo-

genetically and in an order that is, hopefully, logical, I would like to stress that the individual sections may be considered separately as to the validity of the ideas contained in them.

ACKNOWLEDGMENTS

It is customary to leave acknowledgments to the end of most papers. I feel, however, that the ideas presented here have been developed so much out of the stimulation of the work of others and out of so many hours of fruitful discussion with patient listeners that it is only appropriate to state my gratitude at this point.

I would like to thank first my colleagues of the Aphasia Research Section of the Boston Veterans Administration Hospital without whose clinical observations and close criticism this paper would not have been possible, in particular, Dr. Harold Goodglass and Mrs. Edith Kaplan. I would like to express my thanks to two neurologists who grew up under the great German classical tradition, Dr. F. A. Quadfasel, formerly Chief of the Neurology Service of the Boston Veterans Administration Hospital, who (together with Dr. Samuel Tartakoff) first provided the author with the opportunity to study a large aphasic population and who constantly provided able criticism and the benefit of profound knowledge of classical writings on aphasia; Dr. Paul Jossmann, trained in Bonhoeffer's clinic, who provided the author with many invaluable German publications. I owe a special debt of gratitude to Dr. Paul Yakovlev who has read the many drafts of this paper. He has given many hours extending in many cases well beyond midnight to discussing with me the anatomical and broader philosophical aspects of this paper on the basis of his profound knowledge of human and comparative neuroanatomy. In addition, his suggestions as to the organization of this paper have been invaluable; the final arrangement of the material was, in fact, suggested by Dr. Yakovlev and the reader may well attribute much of whatever is lucid to this suggested reorganization.

I would like also to express my gratitude to those of my colleagues, too numerous to mention here, who have given me the benefit of their criticisms of the ideas expressed here; to Professor Oliver Zangwill who urged me to develop these ideas methodically in print; and to Professor Davis Howes who was the principal investigator of the research project under which this

work was done and with whom I have had the privilege of working for several years.

I also owe a considerable debt to the work of others, which I hope is adequately expressed in the following pages. The remarkable set of papers by McCulloch, Bailey, Bonin and their many collaborators were indispensable in providing an anatomical basis for many of the ideas discussed here; the writings of Hugo Liepmann, a surprisingly neglected figure in the history of neurology, provided the outstanding clinical models for thinking in terms of disconnexion. The writings of Dr. Edwin Weinstein were a major influence in directing me to the importance of confabulatory responses.

I would like to express my deep gratitude to my secretary, Mrs. Ceoria M. Coates, who has so effectively dealt with the burdensome task of dealing with the preliminary versions of sections IV–VIII of this paper.

Finally, I should like to express my appreciation for the opportunity of spending several hours with Professor Jerzy Konorski to whom goes the credit for having been the first to return in recent years to an analysis of aphasia from the point of view of intrahemispheric disconnexion; I hope we will soon have the opportunity to see an extensive presentation of his ideas in English.

I. ANATOMICAL BACKGROUND: FLECHSIG'S RULE

The term 'disconnexion syndrome' is applied to the effects of lesions of association pathways, either those which lie exclusively within a single cerebral hemisphere or those which join the two halves of the brain. These syndromes are equivalent to the 'transcortical' or 'conduction' syndromes of older authors. It is appropriate to consider first the anatomical arrangements of such connecting pathways. Flechsig's principle (Flechsig, 1901; Bailey and Bonin, 1951) states that the primary receptive areas (the koniocortices) have no direct neocortical connexions except with immediately adjacent, 'parasensory' areas, the 'association areas' of common neurological usage. As Bailey and Bonin (1951) state this doctrine, "The primary sensory areas do not send messages very far into the surrounding cortex and receive cortical impulses almost exclusively from the parasensory areas; the parasensory areas, on the other hand, receive afferents from several other cortical areas and send their cortico-

cortical efferents much farther away." As stated by Flechsig (1901), "... No long association system is known which connects two primordial zones that are to be regarded as sensory centres...." (By the term 'primordial zones' Flechsig meant regions of very early myelination.)

This doctrine applies not only to connexions within a single hemisphere but also to those between the hemispheres. Thus, the primary visual cortex has no callosal connexions (Myers, 1962a) nor does it have any long connexions to other parts of neocortex within the same hemisphere in the higher primates (Bailey *et al.*, 1950; Krieg, 1963). It has neocortical connexions only with the adjacent concentric association areas 18 and 19. Similarly, although the evidence is less clear, it is probable that the primary auditory and somesthetic cortices project only to immediately adjacent association areas. The limitation expressed in this rule applies only to neocortical connexions. The primordial sensory centres receive fibres from the thalamus and may in turn send long fibres to subcortical regions. The primary visual cortex has, according to some authors, direct connexions to the collicular region but not to neocortex. Crosby *et al.* (1962) express the opinion that even the connexions to the brain-stem in man come exclusively from area 18 while in the monkey these fibres arise from areas 17 and 18, whose borders are, however, harder to distinguish than they are in the human. The visual cortex can thus communicate with other areas of *neocortex* either in the same or the opposite hemisphere only by way of the concentric association cortex.

It is probable that the principle of Flechsig does not hold so strongly for subprimate forms. In the cat Curtis's experiments (Curtis, 1940) showed the presence of direct callosal connexions of the visual cortex while the same author showed their absence in the monkey. Similarly, Yakovlev and Locke (1961) have interpreted some of Cajal's findings in the rabbit as meaning that there is a direct pathway from the visual cortex to the hippocampus in this animal.[2]

These data on the cat and rabbit suggest the general possibility that in subprimate forms association fibres may arise from primary projection areas. With increasing phylogenetic complexity these connexions would be transferred to the newly developed association areas. This would also suggest that the association areas do not appear *de novo* but are elaborations of parts of the projection cortex. It would be interesting to know whether particular elements of the primary sensory cortex migrate out to

develop into more highly elaborated regions, and if so, which elements these might be. This possibility of the differentiation of association cortex out of projection zones is supported by the fact that in man the visual cortex is relatively more condensed than it is in the monkey and lower forms and also by the fact that in the monkey areas 17 and 18 are said by some authors to merge into each other indistintly (Crosby *et al.*, 1962) while in man the border is sharp. It is not inconceivable that some of the more primitive connexions which directly join primordial zones may continue to persist in the higher forms but if so they are probably vestigial and functionally of minor importance. This gradual process of separation of association areas from primary projection areas is probably only the later stage of a process in which the primary motor and projection areas become individuated out of a less specialized brain. Thus, Lende (1963) has recently shown that in marsupials such as the opossum and wallaby, the primary motor and somesthetic areas overlap almost completely, a situation clearly different from the marked if not complete separation in the higher primates and man.

These anatomical facts imply that a large lesion of the association areas around a primary sensory area will act to disconnect it from other parts of the neocortex. Thus, a 'disconnexion lesion' will be a large lesion either of association cortex or of the white matter leading from this association cortex. The specification of the association areas as way-stations between different parts of neocortex is certainly too narrow, but it is at least not incorrect. This view, as we shall see, simplifies considerably the analysis of effects of lesions of these regions. Since a primary sensory region has no callosal connexions, a lesion of association cortex may serve both to disconnect such an area from other regions in the same hemisphere and also to act in effect as a lesion of the callosal pathway from this primary sensory area.

1. *Connexions of the Visual Association Areas*

If the connexions of primordial projection areas are limited to adjacent concentric association areas, we find in turn that the connexions of the association areas are not distributed as widely as one might expect to the remainder of the neocortical mantle. Bonin and Bailey (1947) point out that in the macaque OB (equivalent roughly to area 18) projects most heavily to OB of the opposite side and to areas OA, FC (on the anterior

convexity of the frontal lobe) and TE (lateral and basal temporal lobe) on the same side. Similarly, OB receives afferents from TE on the same side, from the opposite OB, and as we noted earlier from the primary visual cortex (OC). The preoccipital region (OA) has similarly restricted connexions, receiving fibres from OB, TE, and PG (posterior inferior parietal region), with a few fibres from PE (posterior superior parietal region). In a later review of the connexions of the macaque Bailey, Bonin and McCulloch (1950) record that OA projects essentially to the same regions as OB. The results for OA are probably less clear than for OB since its borders are more difficult to define by cytoarchitectural criteria.

Let us consider area OB for the moment since this can be regarded as clearly a visual association area. It has a very restricted afferent and efferent distribution, essentially receiving and sending fibres only to immediately adjacent regions of cortex and establishing major long cortical connexions with a limited number of regions. We may presume that the connexions to area FC relate to motor responses to visual stimulation which we will discuss more fully later on. What then of the only other long connexion of OB, the one to the lateral and basal temporal cortex? This connexion is of course by way of the classical inferior longitudinal fasciculus, a structure well recognized for many years although violent arguments about it went on at the turn of the century. Some authors doubted the existence of this pathway and assumed that all the fibres attributed to it actually were part of the geniculo-calcarine fasciculus. In the monkey it is probably the largest transcortical connexion of the visual association areas. The significance of this connexion of the visual association cortex becomes clearer when we look in turn at the connexions of area TE.

Akert *et al.* (1961) found that when lateral and basal temporal neocortex (TE) and temporal polar neocortex (TG) were ablated in macaque monkeys, secondary degeneration appeared in the white matter of adjacent parts of the parietal, temporal, and occipital lobes; there was also degeneration of a large bundle going to posterior cingulate cortex and heavy degeneration in the uncinate fasciculus. Degeneration was seen in the white matter entering the prepyriform cortex and the amygdala.

Since Akert *et al.* removed both TE and TG, it is important to separate the connexions of each of these regions in primates. Certain earlier studies cited by Akert *et al.* do make these distinctions. Studies on subprimate species probably cannot be considered for reasons already given. Bailey, Bonin, Garol and McCulloch (1943a, 1943b) found that area 38 (temporal pole, equivalent to TG) when strychninized fired only locally. The pole did *receive* afferents from orbital frontal cortex. Petr, Holden and Jirout (1949) working in Bailey's laboratory were able to extend these fragmentary earlier observations by better technique. They now showed that temporal pole sent

fibres to orbital cortex and also fired what they called 'the anterior part of the fusiform gyrus (TH)' and the uncus (H). Other authors would probably name this region part of the hippocampal gyrus.[3] Pribram, Lennox, and Dunsmore (1950) found that strychninization of the temporal pole fired orbital cortex, anterior insula, amygdala and hippocampus. The first three regions are considered very well established connexions of the temporal pole. Segundo, Naquet and Arana (1955) using a variety of techniques confirmed connexions from temporal pole to amygdala and hippocampus, as well as hippocampal gyrus. It seems likely that we can accept these connexions of temporal pole as being well validated.

We may now turn to the connexions of lateral and basal temporal neocortex (area TE, comprising the middle and inferior temporal gyri). The strychninized middle temporal gyrus was found by Petr, Holden and Jirout to fire hippocampal gyrus but only weakly; the inferior temporal gyrus, however, produced significant firing in the hippocampal gyrus. Whitlock and Nauta (1956) found that a lesion of inferior temporal gyrus led to degeneration in basolateral amygdala and in the hippocampal gyrus. In addition, a large connexion was found from the middle and inferior temporal gyri to the dorsomedial nucleus of the thalamus. (Other connexions such as those to the basal ganglia were also found but will not be discussed here.) Poblete, Ruben and Walker (1959) found that after-discharges from TEs pread readily to the homolateral amygdala and hippocampus in the macaque.

Area TE thus projects to the hippocampal gyrus, basolateral amygdala and dorsomedial nucleus of thalamus. The hippocampal gyrus in turn is strongly connected to the hippocampus (Adey and Meyer, 1952). The amygdala has important hypothalamic connexions, as well as connexions to hippocampus via a multisynaptic route (Gloor, 1960) and a large projection to the dorsomedial nucleus of the thalamus (Nauta, 1962). The dorsomedial nucleus of the thalamus in turn has important hypothalamic connexions (Crosby *et al.*, 1962).

In none of these studies has there been any mention of connexions to the posterior cingulate region as mentioned by Akert *et al.* (1961) and Bucy and Klüver (1955). At present it would not be possible with assurance to assign this to the lesion of TG or to that of TE. It would appear likely that the temporal polar lesion is most likely the responsible one since the temporal pole may well make up part of the ring formed by the cingulate and hippocampal gyri and anterior insula (Kaada, 1960).

Let us summarize the conclusions from these studies. In order to understand better the reason for the large projection from the visual association cortex to area TE, consisting in the monkey of the middle and inferior temporal gyri, we have looked at the projections which in turn leave TE. The data support the notion that the connexions of TE are to limbic structures which in turn have important connexions with hippocampus and hypothalamus, i.e. with parts of the central core intimately involved in learning and in emotional responses. Thus TE (particularly the third temporal gyrus) projects (1) to hippocampal gyrus which in turn projects strongly to the hippocampus; (2) to amygdala which has important direct connexions to the hypothalamus and to the dorsomedial

nucleus of the thalamus, and important indirect connexions to the hippocampus; and (3) directly to the dorsomedial nucleus of the thalamus which in turn has important hypothalamic connexions. Akert *et al.* (1961) in the conclusion of their paper on the effects of the removal of temporal neocortex in the macaque stated, "The significance of this study seems to lie in the further demonstration of close functional and anatomical relationship between rostral association cortex of the temporal lobe and the rhinencephalon." I would agree with this conclusion but with the important addition that this is true even if one confines one's attention to area TE alone without taking the temporal pole into consideration.

We have thus far described the pathway which proceeds from visual cortex to visual association areas 18 and 19, thence to lateral and basal temporal lobe and from there to limbic structures. Let us consider briefly the pathways in the reverse direction.

Votaw (1960) studied the degeneration which followed hippocampal lesions in the monkey; in addition to the expected changes in the fornix, he found "a definite and important hippocampotemporal path, discharging from the hippocampus to the hippocampal gyrus and to other gyri of the temporal lobe...." Votaw explains the functional significance of this projection by suggesting that "the hippocampus projects to the temporal lobe where it is known that motor responses of an extrapyramidal nature can be obtained...." While not disagreeing with this interpretation, I will propose later that these connexions may be part of a pathway from hippocampus to visual association areas having functions other than, and perhaps in addition to, those suggested by Votaw. Gloor (1960) has reviewed the connexions of the amygdala very extensively. He notes that its cortical projection field is very limited in being restricted to hippocampal gyrus (pyriform area), temporal pole and insular cortex. The hippocampal gyrus in turn probably has connexions to temporal neocortex. It has important connexions with the hippocampus but probably via a polysynaptic route. He also finds no evidence for efferent cortical connexions of this structure beyond the confines of the temporal lobe. While the uncinate fasciculus if often mentioned as a connexion of the amygdala, it is likely that it actually connects basal and medial cortex of the temporal pole to the frontal orbital region. The amygdala receives an afferent connexion from dorsomedial thalamus although the connexion in this direction is not as large as that in the opposite sense (Nauta, 1962). The amygdala may thus act as a way-station from this part of the thalamus to the temporal lobe.

We may say in summary that although knowledge of projections *to* the temporal lobe *from* limbic structures is less complete than is our information about those projections going in the opposite direction, it appears that the hippocampal gyrus certainly receives afferents from limbic structures and that it may perhaps act as a relay from these structures to temporal neocortex. Further study of the connexions of the hippocampal gyrus may

further elucidate this problem. We can probably regard portions of the temporal neocortex as perhaps representing association cortex for the limbic structures just as areas 18 and 19 represent visual association cortex. The concept that lateral and basal temporal neocortex and temporal pole are to be regarded as the association cortex of the limbic system is further strengthened by the fact that it is precisely these areas of the temporal lobe which utilize the anterior commissure rather than the callosum for their connexions to the opposite hemisphere. In fact Akert *et al.* (1961) summarize the regions ablated in their experiments as "non-callosal temporal cortex." They found no trace of callosal degeneration in their experiments. The fact that these parts of the temporal lobe utilize a commissure strongly associated with the limbic system is a further evidence of their close functional relationship to the limbic system and is at least compatible with the idea that they can be regarded as the association cortex of the limbic system. They are thus contrasted with the association cortex concentric to the primary projection areas which makes use of the corpus callosum for connexions to the opposite side.

What is the significance of this two-way connexion from visual cortex to the limbic system? I will now present a theory of the functions of these pathways which I believe is supported by the published experimental investigations of animals with lesions in these connexions. The next section will, after a brief introduction, summarize some of these experiments and present a theoretical interpretation of the findings.

II. AGNOSIAS IN ANIMALS

The study of lesions of the association cortex itself or of the connexions of this cortex leads naturally to a consideration of a group of disturbances in animals, at least in the primates, which may be reasonably described as *agnosias*; for the moment these may loosely be defined as disturbances of recognition in the presence of intact elementary sensation. As we shall see, the evidence is great that such disturbances do occur in animals; I will attempt to explain their mechanisms by reference to the anatomical arrangements of the association areas and their relations to limbic structures. In a later section I will discuss agnosias in man and will present the thesis that the mechanism for human agnosias is probably different from the mechanism of those seen in primates. Although the order of presenta-

tion of the data makes, I feel, evolutionary sense, I would like to stress again that the interpretation of the human syndromes can be considered independently of the animal material.

1. *Removals of Temporal Neocortex: the Visual-Limbic Disconnexion Syndrome*

Klüver and Bucy (1938) showed that removal of the temporal lobe (neocortical and limbic structures) led to a characteristic syndrome which prominently included loss of the ability to make correct choices under visual control.[4] This disturbance occurs in the presence of many evidences of excellent retention of vision (Klüver and Bucy, 1938; Pribram, 1962), which led Klüver and Bucy to speak of the disturbance as a visual agnosia. Later investigators found that such disturbances in visual choice could be reproduced by lesions involving the temporal neocortex, in particular the middle and inferior temporal gyri, i.e. area TE. I will not cite here the many studies which have contributed to the clarification of this problem but would refer the reader to the paper by Chow (1961) where much of the relevant literature is reviewed by one of the major contributors to the investigation of this problem. The general conclusion of these studies has been that excisions of area TE bilaterally lead to a loss of previously acquired learning to choose one of two complex visual stimuli. These monkeys can relearn these tasks and can learn new visual choices but usually more slowly than normals. Furthermore, these delimited temporal lesions do not produce defects in learning in other modalities, nor do cortical lesions elsewhere in the brain produce such disturbances with visual tasks.

Why should the lesion of TE produce such a disturbance? Looking at exactly how the monkey learns to make the choice will help to make the reason clear. In order to highlight the mechanism, I will first present a procedure that is slightly different from that generally employed. Assume the monkey to be presented visually with a cross and a circle. If he presses on either figures a small pellet comes out of a chute beneath that figure. The pellets are identical in external appearance. The one beneath the cross turns out, however, to consist of a food that is normally fed to monkeys, while the pellet released on pressing the circle is inedible, perhaps through admixture of some bitter component. We would find that the monkey will soon learn to press the cross and receive the edible

pellet and to avoid pressing the circle which would lead to his receiving an inedible pellet. He must somehow learn to associate 'cross' to 'edible', 'circle' to 'inedible', i.e. to make a visual-gustatory or visual-olfactory association, or more generally a visual-rhinencephalic association (where 'rhinencephalon' is used in the narrow sense). These olfactory or gustatory structures lie in the central core of the brain. I have suggested earlier that the pathway from the visual cortex to these rhinencephalic structures is via the lateral temporal neocortex whose destruction leads, therefore, to a disconnexion between the visual and rhinencephalic regions.

We may widen our view somewhat by noting that most of the stimuli which act as positive reinforcements in learning experiments, whether food, water or sexual objects, seem to depend on systems to which the structures of the medial temporal region have rich connexions. We may thus look at the lesion of lateral and basal temporal lobe as leading to a 'disconnexion from reinforcement' without specifying the modality of the reinforcement.[5]

We can consider the data from still another point of view that is probably not fundamentally different but rather complementary. Konorski (1961) regards lesions of association areas as producing modality-specific deficits in recent memory, basing his conclusions on experiments performed by himself and others. I will not comment on his interesting studies on frontal association cortex but will confine myself to his discussion of lesions of the visual and auditory association areas. His interpretation is that the projection areas and association areas form a reverberating circuit which is destroyed by lesions of the association areas. He ends his paper by briefly mentioning the effects of hippocampal lesions which produce defects of both auditory and visual recent memory in monkeys. He concludes "The physiological mechanism of these deficits (i.e. those resulting from hippocampal lesions) seems to us so far not clear and they require more detailed investigation."

I believe that the view which I have advanced above fills in the gap in Konorski's system by providing the nexus between the effects of lesions of association areas and lesions of the hippocampal region: the important fact is that the sensory association cortex projects to the medial temporal structures and receives projections from them. The hippocampal region thus probably communicates with all sensory modalities. The appropriate

association area lesion may specifically disconnect the hippocampal region from a single modality and produce a recent memory defect in that modality alone. Konorski's theory of a reverberating circuit should therefore be amplified to include the hippocampal region.

I have not discussed here Dr. Konorski's interesting theory of the functions of the frontal association cortex. In later sections of this paper where the role of the motor cortex is discussed I will revert to the question of the functions of the association cortex lying immediately anterior to the classical motor cortex. The problem of frontal lobe associative functions is made more difficult by the fact that some portions of the human frontal lobe are extremely advanced phylogenetically and may represent new evolutionary developments. Comparisons may be difficult even between lower primates and carnivores. It is, of course, likely that the frontal lobe contains several association regions of differing function.

Dr. Konorski has stressed the role of the frontal lobe in inhibitory conditioned responses. It is possible that the orbital cortex with its extensive connexions to limbic structures via the uncinate fasciculus and the temporal pole may be exerting a predominantly inhibitory influence on limbic reactions. Egger and Flynn (1963) have shown that some portions of the amygdala (in cats) inhibit hypothalamic attack behaviour while others facilitate it. It would be interesting to study the cortical connexions of the inhibitory and facilitatory regions. It would be most interesting to see whether the inhibitory regions are receiving their main connexions from the orbital cortex via uncinate fasciculus and temporal pole. Other parts of the frontal lobe have important connexions to the cingulate gyrus as has been stressed by such workers as Showers (1959) and Nauta. The significance of these connexions is still to be determined. The role of connexions to the hippocampal region from the frontal lobe in motor learning and particularly in inhibitory learning also deserves further study.

Certain other aspects of the syndrome produced by lateral and basal temporal neocortical ablations are worthy of comment here. At one time the view was common that the Klüver-Bucy syndrome was the effect of a mixture of independent temporal neocortical and rhinencephalic disturbances. I have already suggested that the visual learning disturbances are the result of disconnexion of the pathways between the visual cortex and the rhinencephalon. A bilateral lesion of the hippocampal region produces a learning deficit as Scoville and Milner (1957) showed in humans. Stepien, Cordeau and Rasmussen (1960) have shown that bilateral ablations of the uncus, amygdala, hippocampus and hippocampal gyrus in monkeys led to a recent memory deficit for both vision and audition. It seems reasonable to interpret the isolated visual recent memory deficit that many investigators have shown as a sequel to removals of area TE as the effect of destroying the connexions of a specific modality to these medial structures. Can other aspects of the Klüver-Bucy syndrome also appear in animals

with neocortical ablations as a result of disconnexion of a single modality from limbic structures?

Most investigators have not recorded finding many of the other disturbances found by Klüver and Bucy. However, Akert, Gruesen, Woolsey and Meyer (1961) have found some of these disturbances in their monkeys with neocortical temporal ablations. These investigators removed temporal pole (TG) as well as lateral and basal temporal cortex so that the final interpretation of their results must await further study. It may be instructive, however, to look at their experiments from the point of view being expressed here.

The monkeys operated on by Akert *et al.* showed marked diminution in emotional responsiveness in certain situations. They showed a markedly diminished fear response to brooms, nets, hoses and toy snakes. It would seem reasonable to explain this by the fact the lesion had effectively cut much of the pathway between the visual cortex and the limbic system, so that arousal of limbic responses by visual stimuli no longer occurred. A piece of evidence that is in support of this idea is the observation of the authors that "The monkeys, throughout their post-operative career, reacted violently only to physical restraint; they would bite if held down firmly but would do so with substantially less vigour than the normal animals." It would thus appear possible that the tameness was most marked to visual stimuli; this would be in keeping with the idea that the lesion was effecting primarily a disconnexion between visual cortex and limbic system.

The possibility of the existence of modality-specific tameness or loss of fear is supported by the findings of Downer (1962). He destroyed the amygdala unilaterally in monkeys combining this operation with section of the optic chiasm, corpus callosum, and anterior commissure. These monkeys were tame in response to visual stimulation when it was confined to the amygdalectomized hemisphere but were normally aggressive in response to visual stimuli confined to the normal hemisphere. These animals, however, responded with aggressiveness to tactile stimulation of either hemisphere. Their tameness was therefore confined to visual stimulation of the amygdalectomized hemisphere. It should be added, parenthetically, that this experiment also confirmed other views that amygdalectomy generally leads to tameness (e.g. Pribram, 1961). We would assume that the tameness of the animals of Akert *et al.* to visual

stimulation may have been due to disconnexion of complex visual stimuli from the amygdala. Whether all the changes in emotional responsiveness depended on this disconnexion or on disconnexion from other limbic structures remains a subject for further study.

Akert *et al.* observed no changes in sexual behaviour. Their monkeys, however, were juveniles. The possibility must be considered that in more mature monkeys with similar temporal neocortical lesions the pattern seen by Klüver and Bucy of indiscriminate mounting of other monkeys, regardless of sex, and even of inanimate objects, might occur. The hypothesis in this case would state that since the limbic system is disconnected from visual cortex, the monkey might not be able to use the visual system to control the patterned sexual activities which arise from limbic activity. One can think of this situation as one in which, as far as the limbic system is concerned, the monkey is blind and he selects possible sexual objects randomly as might a blind monkey who (for other reasons) cannot use his visual learning to guide appropriate sexual activity.

The monkeys of Akert *et al.* did not eat ground meat, unlike the Klüver-Bucy monkeys. One possibility is that this behaviour resulted not from visual-limbic disconnexion but was the result in Klüver and Bucy's experiments of direct damage to rhinencephalic connexions so that there was a diminution of olfactory or gustatory sensitivity. This problem deserves further investigation.

Klüver and Bucy pointed out that a normal monkey will place an inedible pellet in his mouth but rapidly learns not to pick it up; by contrast their temporal lobectomized monkeys would repeatedly pick up such pellets, place them in their mouths and then reject them. Although the description is not given in detail, it appears that the animals of Akert *et al.* showed this behaviour in attenuated form. The explanation for this phenomenon would also follow from the fact that the visual cortex is disconnected from the olfactory and gustatory cortex and that therefore the visual cortex can never 'learn' that an object it sees is inedible.

The behaviour described as release of oral tendencies, i.e. the exaggerated tendency of the animal to take objects in his mouth is another feature that the animals of Akert *et al.* shared with Klüver and Bucy's animals. It appears possible that this also results from the failure to acquire visual-rhinencephalic associations. The animal now uses the retained pathway via oral sensation for ascertaining whether the objects

are edible. This behaviour might therefore be analogous to the tendency of the blind to palpate objects and might free us from the need of assuming a regression to an 'oral' stage of behaviour.

I have dwelt at some length on these experiments on neocortical temporal lesions and their interpretation since I feel they illustrate the principles underlying the anatomical organization of the association areas in sub-human primates. It will be useful to discuss here some aspects of this problem and then go on to some possible objections to the theory,

2. *The Effects of the Extent of the Lesion*

I would like to lay stress first on the importance of the extent of the lesion. Thus Meyer (1958) points out that in the study by Akert *et al.* certain aspects of the Klüver-Bucy syndrome were seen which were not observed by Chow who had performed comparable but less extensive ablations of temporal neocortex. Meyer comments, "This (*sc.* difference in results) may be due to a difference in the size of the lesions for smaller ones characteristically are not sufficient." The importance of the size of the lesion is probably the same for all disconnecting lesions, i.e. for lesions of association cortex or of fibre connexions of association cortex. There is little evidence to suggest that very discrete lesions of association cortex or of highly delimited fibre pathways of limited extent have major behavioural effects. There is thus at least a definite quantitative difference between lesions of association areas and of primary projection areas, where even small lesions tend to produce definite effects. This may well be a qualitative difference and suggests a fundamental difference in organization.

The importance of size suggests that there is an important degree of equipotentiality in the association cortex and its connexions. Several experiments point to this conclusion. Thus, Ades and Raab (1949) point out that extensive destruction of the visual association cortex[6] in the macaque led to loss of a previously learned visual discrimination. But, if the operation was done in stages with practice between the serial ablations the task was retained. Similarly the task was relearned after bilateral simultaneous extirpation of these pre- and para-striate regions but lost again after an excision of the lateral and inferior temporal region which spared the temporal pole.

Another characteristic of these lesions which suggests equipotentiality is

the sensitivity of the test procedures to complexity. Ades and Raab's monkeys who failed to learn a form-discrimination could learn a black-white discrimination. Similarly, as Meyer (1958) points out, in the experiments of Akert *et al.* monkeys who eventually were able to relearn form discriminations could not learn a more complex visual task involving the formation of discrimination learning sets. A related experimental result is that of Orbach and Fantz (1958) who found that overlearning before operation prevented loss of form-discrimination after ablations of the temporal neocortex.

The above experiments all seem to support the picture of the visual association cortex proper and area TE as forming an equipotential area with learning being diffusely represented. Overlearning leads to high redundancy and consequently little loss after partial ablation. Simple tasks can be learned after partial ablation but not more complex ones which require a greater expanse of cortex. The possibility is certainly raised that even in relation to simple tasks certain defects might be shown with appropriate testing, e.g. slowness in learning to reverse a discrimination, or the presence of marked interference effects (i.e. poorer retention of a task when similar tasks are interposed between testing sessions) which would appear to be reasonable consequences of cutting down the total amount of cortex involved. Riopelle and Churukian (1958) have, in fact, made a careful study of the problem of interference effects in monkeys including a group with extensive bilateral ablations of the lateral surfaces of the temporal lobes. While this group did very poorly in learning to make visual choices, they showed no evidence of such interference effects as I have postulated. I believe, however, that it might still be useful to press further the study of the relationship between extent of lesion and the magnitude of interference effects.

This very question of equipotentiality raises certain problems. We have implicitly assumed that the connexions from visual cortex via visual association areas to lateral-basal temporal neocortex and thence to limbic structures are exclusively devoted to visual-limbic associations. We have thus implicitly excluded the possibility that some components of this system are in fact not modality-specific and thus could make up part of the pathway from other primary sensory areas to rhinencephalic structures. Further experiments to be presented shortly in more detail point clearly to the fact that there is at least a gross separation of the pathways

by modality. The possibility of significant overlap is not entirely excluded by these results but complete equipotentiality of all association systems can be rejected.

3. *Non-Limbic Associations*

A second implication of the above discussion follows from the fact that the major connexions of the visual region feed eventually into limbic structures. It would appear therefore that non-rhinencephalic associations should not be of much importance in the monkey. Therefore, while visual-limbic associations are readily formed, bonds between vision and other modalities should be weak. This conclusion is compatible with many results particularly in conditioning theory on the weakness of sensory-sensory links and on the difficulty of chaining conditioned stimuli. Reinforcement cannot be too remote and reinforcement depends on rhinencephalic activity. As I have noted earlier, it is probably not correct to make the general statement that sub-human forms have difficulty in forming cross-modal associations since they, in fact, readily form associations to 'limbic' modalities, such as, pain or olfactory-gustatory sensation; by contrast they perform poorly in establishing links exclusively between the non-limbic modalities (audition, vision, and somesthesis). Such purely 'non-limbic' associations seem to be formed readily and stably only in man under certain conditions. Burton and Ettlinger (1960) suggest "that language may serve as a cross-modal bridge under these... conditions"; in other terms they are suggesting that 'verbal mediation' is the means by which humans achieve cross-modal transfers. This theory, however, in my opinion, evades the fundamental point, to which we will return again, *that the development of speech itself depends on the ability readily to form stable intermodal associations*, particularly visual-auditory and tactile-auditory bonds. I have thus, as it were, inverted Burton and Ettlinger's statement: I would argue that because man can form certain intermodal associations, he can develop speech; once he has developed speech, he can succeed in turn in forming other intermodal associations.

Wilson and Wilson (1962) have pointed out that prior experience with 75 tactual object-choice situations facilitated visual-learning set acquisition when their test group of normal monkeys was presented subsequently with 75 different visual discriminations. However, as the authors themselves comment, "The present results do not throw any light upon the question of

whether intermodality transfer of specific discriminations is possible.... However, the existence of general transfer of the kind here demonstrated must be borne in mind in planning experiments on specific transfer." I would agree with their view that these general effects may well depend on such factors as the elimination of error tendencies rather than on those factors involved in the transfer of information about a specific problem between modalities.

The problem of cross-modal learning deserves further study and the recent interest in this area is most welcome. It should be pointed out that experimental design is of vital importance here. Many experiments in the literature are really tests of the ability of the animal to respond to multimodal stimuli and not necessarily of his ability to form intermodal associations. Thus, the ability to respond to both a sound and a light but not to a sound alone need not depend on visual-auditory connexions but might require only parallel visual-limbic and auditory-limbic associations. It would seem to me that the most reasonable type of experimental design in which cross-modal transfer was obligatory would be that using sensory preconditioning (Hilgard and Marquis, 1961). Here the animal is first exposed without reinforcement to, let us say, a visual stimulus followed by an auditory one. Later on he would receive reinforcement following the auditory stimulus presented alone. Finally, he would be presented with the visual stimulus alone. Appropriate response to the visual stimulus (proper controls having been instituted to rule out nonspecific generalization) could then result only from the previous establishment of a visual-auditory bond.

Do the anatomical findings show no basis for cross-modal response? Unfortunately the exact anatomy of the interconnexions between association areas has hardly been worked out in detail. The experiments of Sugar, French, and Chusid (1948) provide some answer. They found (as we would expect from Flechsig's principle) no connexions between primary visual and primary auditory cortex. They found, however, connexions from auditory association cortex in the supratemporal plane to the anterior wall of the lunate sulcus (i.e. part of area 18) as well as some connexions to the exposed surface near this sulcus. Connexions in the reverse direction from visual association cortex to auditory association cortex are much less powerful. Sugar *et al.* state that their results confirm earlier results of Mettler's based on anatomical findings.

These results suggest that there is apparently some basis for auditory-visual associations, and probably for much weaker associations in the reverse direction. The connexions present are probably not extensive (they are certainly far less massive than those from pre- and para-striate regions to the lateral and basal temporal lobe), but it certainly would be desirable to explore further their physiological significance. They probably constitute the basis for weak non-limbic intermodal associations. As I will point out in a later section, it is likely that cross-modal associations involving vision, audition, or somesthesis (in contrast to, let us say, the visual-limbic, tactile-limbic, and auditory-limbic associations of sub-human primates) become prominent only in man, and that it is probably associations involving the auditory system which are most significant in the development of language. As I shall also suggest later, there is some evidence for the existence of an extensive anatomical substrate which can subserve a much larger number of such associations in man than in sub-human primates.

4. *'Motor' Learning*

Another aspect deserving some comment here is that of the motor responses involved in learning. Thus, if the monkey reaches out and presses a cross under which there is a peanut it can be argued, as we have done, that he has learned a visual-gustatory association. But does not his motor response also suggest that he has learned a visual-motor connexion? I will discuss the problem of visual-motor learning in more detail in the section on the apraxias but some clarification appears reasonable here. In the first place many motor responses under visual control may not depend at all on connexions between visual and motor cortex. Thus, rage responses to visual stimulation probably depend on a pathway proceeding from visual cortex to visual association cortex, thence to lateral and basal temporal lobe and finally reaching the limbic system and here triggering off highly patterned behavioural sequences, mostly probably innate. Thus MacLean (1960) points out that Hess and Hunsperger obtained 'full-blown angry behaviour' in the intact animal by stimulation of appropriate regions either in the hypothalamus or the central grey of the mid-brain. Other experimenters such as Bard had shown that highly patterned rage reactions were dependent on the intactness of connexions of the hypothalamus. It is thus not necessary to depend on connexions to motor cortex from visual cortex for such behaviours.

Furthermore, other motor reactions may also depend on pathways descending to the brain-stem from association areas. Large movements particularly will result from stimulation of supplementary motor areas projecting directly to brain-stem structures (Crosby *et al.*, 1962). It is true that even single limb movements are obtainable from these areas but these are usually much grosser than the delicate movements obtainable from the classical motor cortex. The important studies of Voneida (personal communication) strongly suggest that some visual-motor tasks are performed via pathways going from the cortex to the brain-stem.

It would seem quite likely that for either highly patterned emotional behaviour or for gross movements in response to, let us say, a visual stimulus, no connexions are needed from visual association areas to the frontal motor cortex. It would seem likely that involvement of motor cortex becomes necessary in relation to the learning of fine movements (e.g. finger movements particularly, precise reaching, or those movements of large muscles involving a very small number of motor units). Another possibility, suggested by the necessity of some parts of the frontal region for delayed-response learning, is that visual-frontal connexions might be brought into play when the motor response to a visual stimulus must be delayed. The recent work of Glickstein *et al.* (1963) is consistent with this view. In any case the term 'visual-motor' may well correspond anatomically to a variety of different mechanisms, of which only one or two involve cortico-cortical connexions. Presumably in man cortico-cortical connexions are more important in motor learning, a point to which I will return in the discussion of the apraxias. Even in man some responses to stimulation probably depend on pathways descending directly to the brain-stem, as I will point out in discussing the preservation of whole-body movements in some patients who show apraxic difficulties with individual limb or face movements.

In all cases of visual-motor learning except the one where the motor response is a 'limbic' motor response, it is necessary to ask what the exact relationship is of the limbic structures to the learning. Thus, when a delayed motor response is carried out with a visual stimulus, are both visual-limbic and frontal-limbic connexions involved? Or can this learning take place with retention only of frontal-limbic connexions? As Riopelle and Churukian (1958) point out most experimenters have concluded that temporal lesions did not lead to difficulties in delayed response. More

complex experiments could, however, be devised to study the question of whether multiple paths to limbic structures are involved in some types of learning.

5. *Objections to the Theory*

One objection that warrants brief consideration is the one that lesions of lateral and basal temporal neocortex lead to simple 'perceptual' disturbances and that to treat them as 'associative' or 'memory' disturbances is incorrect. Many lines of evidence militate against this view. Thus, Klüver and Bucy (1938) point out that their animal (which, of course, had a temporal lobectomy, a procedure more extensive than the lateral and basal decortication which I have been discussing) promptly picked up fragments of white peppermint, some as small as a fraction of a millimetre from a black table or very small pieces of banana from a white background. Bucy and Klüver (1955) mentioned the expertness of their operated monkeys in catching cockroaches. As Pribram (1962) has indicated, these animals are capable of catching flying insects.

Some authors have concluded that infero-temporal lesions may have effects on visual acuity (Pasik *et al.*, 1960). Animals with such lesions may show difficulty in discriminating small differences in size. It is, however, clear that animals with subtotal striate cortex lesions, which produce a much greater increase in the size discrimination threshold, have much less difficulty in discriminating painted patterns than do the infero-temporal monkeys (Wilson and Mishkin, 1959). These authors believe, however, that the striate lesion produces some deficit of learning and that the infero-temporal lesion produces some 'sensory' deficit since the first group did show some impairment in painted pattern discrimination while the infero-temporal group did show some loss in visual acuity. I would feel, however, that the dissociation is more complete than these authors are willing to concede. They themselves point out that the learning deficits of the striate lesion group may well be simply related to sensory deficit, particularly in colour vision. Secondly, the fact that the infero-temporal operates showed a more rapid diminution in performance on a size discrimination test as the difference in size between the test objects diminished does not prove that they had acuity defects. Any difficulty in learning is multiplied by increasing the difficulty of discrimination. A native English speaker who understands normally spoken English and French

will almost certainly show a more rapid rate of decline in his comprehension of French as the rate of speech goes up than he will show in his comprehension of English. Viewed from the anatomical point of view a severe enough lesion of the striate cortex must eventually limit the information reaching the association cortex; a large enough lesion of association cortex may eventually limit accurate handling of what comes from the striate cortex. In this sense Wilson and Mishkin are correct in expecting some overlap of impairment. This view is, however, compatible with markedly different organizations of primary visual and associative cortex.

6. *Negative Experiments*

The most serious problem of all for the theory is posed by certain negative experiments. According to the view proposed here the outflow from the visual cortex is by way of the visual association areas to the lateral and basal temporal neocortex. This implies that lesions of the visual association areas 18 and 19 should themselves produce the same picture. Ades and Raab (1949) showed that monkeys lost a visual form discrimination after extirpation of the visual association areas but could later reacquire it. Chow (1961) has made a long and careful study of the visual association cortex. In his latest studies he found that in one monkey ablation of parastriate cortex (to which a pulvinar lesion was also added) led to loss of a visual choice learned pre-operatively although the animal reacquired the task in as few trials as before ablation; the second animal with the same lesion showed considerable savings in relearning this task. Both animals showed savings in relearning a choice of vertical versus horizontal striations (although the second animal showed more savings than the first). But these effects were less marked than those of removing temporal neocortex or of cross-hatching the temporal cortex.

These experiments raise two problems. Why are the effects of removing the para- and peri-striate regions not as profound as those of removing temporal neocortex and why in all the experiments we have cited is there so much ability to reacquire form discriminations? I suspect that the answer lies at least partly in incompleteness of lesions. Chow (1961) himself pointed out that he had never succeeded completely in removing the parastriate areas. This difficulty is increased by the fact that in the monkey

the borders of areas 17 and 18 are perhaps less clearly demarcated than they are in man (Crosby *et al.*, 1962). In addition there is particularly great difficulty in removing the association cortex on the medial surface. Ades and Raab (1949) specifically point out that "the medial and posteroventral parts of areas 18 and 19 usually escaped serious invasion because of their inaccessibility." Their lesions also did not extend farther posteriorly than the posterior bank of the lunate sulcus and therefore some association cortex was almost certainly spared. Is there any way of ensuring complete removal of association cortex? It is possible that physiological criteria may be the most useful means of determining the appropriate extent of surgery. Thus, one could remove all that pre- and para-striate cortex which when strychninized gives responses in the lateral and basal temporal lobes.

There is yet another possibility to consider in the attempt to reconcile the general lack of marked results of pre-striate ablation with the known anatomical facts. Is it conceivable that certain connexions which are not active in the normal animal and hence do not respond to strychninization become active as the result of prestriate ablations? Is there, as it were, an unused reservoir of connexions proceeding directly from the striate cortex to the lateral temporal neocortex? This need not be merely a speculation but could be studied experimetnelly. After determining the pattern of response to strychninization of a normal macaque brain, one could perform pre-occipital removals and at a later date study the pattern of responses to strychninization to see if there is evidence of the opening up of previously unused pathways.

Could the important pathways from striate cortex to temporal lobe involve synapses in subcortical structures? Chow (1961) concludes from his own experiments (in which cross-hatching the temporal lobe led to effects similar to those of temporal ablations) that probably cortico-cortical connexions must be implicated and not connexions via the thalamus. Jasper *et al.* (1952), however, found that after-discharges spread from the striate cortex to the pulvinar and superior colliculus (possibly by way of area 18) but not to parastriate cortex (area 19). Parastriate cortex itself also projected strongly to the thalamus. It would appear that the problem of the negative results of most studies on pre- and parastriate ablation deserves extensive further study both physiologically and anatomically.

7. *Other Reward Systems*

In the experiments I have so far cited the monkeys were rewarded with food. Since I have laid great stress on the interpretation of these disturbances as disconnexions between visual and rhinencephalic systems, the question must naturally arise as to whether the temporal neocortical lesions effectively disconnect the visual system from all reinforcements. I suspect that this is the case; there are, however, important reasons for investigating this problem. Could learning in response to pain be preserved in animals who have undergone resection of lateral and basal temporal neocortex, despite the loss of learning in response to positive reinforcers? Furthermore, would such a discrepancy reflect only the greater effectiveness of pain as a reinforcer or might it reflect anatomical differences in the pathways involved?

Of even greater interest would be the use of less obviously 'limbic' reinforcers. Thus, Butler (1953) has shown that monkeys will learn in order to have the opportunity to view briefly the environment outside the cage. He points out that this cannot be regarded as a 'secondary' reinforcement since second-order conditioned responses extinguish rapidly, while the criteria of learning in these experiments remain stable over long periods of time. Does this type of learning also depend on connexions between the primary sensory areas and the limbic system mediated by way of the association areas? Or is this type of learning 'self-reinforcing', in the sense that it does not need limbic connexions? The fact that animals with the Külver-Bucy syndrome still show a high degree of curiosity in examining objects visually suggests the possibility that this type of reinforcer might still be effective after removal of temporal neocortex. The possibility even exists that some of the recovery in animals with these ablations depends on the preservation of this variety of reinforcement. It is evident that the results of an investigation of this problem would be of importance.

8. *Lesions of Somesthetic Association Areas*

Blum (1951) demonstrated disturbances of somatosensory discriminative behaviour with lesions involving the parieto-temporo-preoccipital region. Pribram and Barry (1956) were able to demonstrate that monkeys with similar ablations (extended, however, to involve the medial surface of the

superior parietal lobule) showed a decrement of retention of a somesthetic task, with no impairment in a visual task. More recently Ettlinger (1962) and Ettlinger and Kalsbeck (1962) have demonstrated the effects of unilateral posterior parietal ablations in monkeys. They found three effects of this procedure which I will attempt to interpret. These consisted of impairment in acquiring a tactile discrimination with the contralateral hand, a failure of transfer of learning from the ipsilateral to the contralateral hand, and a disturbance of visual reaching with the contralateral hand. I will try to show that the type of analysis given in detail for the visual system applies equally well to the somesthetic system.

It is important to specify the extent of the ablations in these animals. In one report Ettlinger and Kalsbeck (1961) describe the regions involved as "what used to be called areas 5 and 7." However, the lesions clearly extend beyond these regions and are more accurately described in the paper by Bates and Ettlinger (1960) as "superior parieto-occipital ablations." An even more complex description would be preferable since the removals involved areas PE and PG posteriorly in the parietal lobe, area OA and the most posterior portion of the superior temporal gyrus (TA). Thus, in addition to areas 5 and 7, area 19 was extensively involved and perhaps part of area 22.

In these experiments Ettlinger and Kalsbeck rewarded the monkey with food if he made the correct choice in a shape-discrimination test. We may apply to this problem the same type of analysis used in the discussion of visual choice experiments. What is demanded of the animal is to make a somesthetic-rhinencephalic association, i.e. to associate a particular shape to the food reward. We must therefore concern ourselves with the pathways from the primary somesthetic cortex to the rhinencephalon.

We would not be able to exclude the existence of a pathway descending from the parietal lobe via the external capsule. Most of the evidence, however, is against this (Crosby *et al.*, 1962). Thus, we find that the external capsule carries fibres running to the basal ganglia from cortical motor areas other than the precentral gyrus. The only large group of cortico-cortical fibres in this system consists of connexions passing in an antero-posterior direction. We must therefore look for another path from the somesthetic cortex to the medial temporal region. Sugar, Amador and Gripponissiotis (1950) studied the connexions of PB (area 3 of Brodmann) forming the posterior wall of the central sulcus. They found that this area

except for some weak connexions shares a common pattern of relationships with PC (area 1) which forms the exposed surface of the post-central gyrus. Both regions respond to peripheral sensory stimuli and receive afferents from the nucleus ventralis posterior of the thalamus. Neither area has significant callosal connexions. Both project most heavily to immediately post-jacent parietal regions PE and PG. Both send connexions anterior to the central sulcus, but these connexions, at least for PB, are weaker than those just mentioned to the posterior parietal region. These two regions therefore appear to have no significant long connexions either within or between the hemispheres and must therefore depend for their long connexions on adjacent regions of association cortex in the posterior parietal region.

According to Bonin and Bailey (1947), PE in turn projects to PC, PG, and FA. The posterior part of TE receives fibres from PG, the posterior inferior parietal region. It would thus appear that while there are no direct connexions from somesthetic association cortex to temporal lobe, there is an indirect pathway to temporal neocortex which travels around the posterior end of the Sylvian fissure. As we have noted earlier, there are connexions from temporal neocortex into the limbic structures lying along the medial surface of the temporal lobe. It would be most useful, however, to have a more detailed knowledge of the pathway from somesthetic cortex to these limbic structures and to see to what extent this is separable from the corresponding pathway of the visual system.

It would therefore be my speculation that the lesions in Ettlinger and Kalsbeck's experiments acted to disconnect the somesthetic cortex from the limbic system and thus led to much the same series of learning and retention difficulties as were caused by a similar disconnexion involving the visual system. The difficulty in making tactile choices may be regarded as resulting from failure to form tactile-gustatory associations; more broadly we can regard it as a disconnexion from reinforcement. In still other but essentially equivalent terms, we may, following Konorski (1961), speak of a recent memory defect specific to the tactile system; we would regard this as resulting from isolating somesthetic cortex alone from the hippocampal region.

The second finding of interest to us in these experiments is the failure of transfer of tactile learning between the hands. Ettlinger (1962) points out that unilateral posterior parietal ablations on either side prevent

transfer of a tactile discrimination learned with the left hand to the right hand. The interpretation of this result follows from the same anatomical principles I have already discussed. As I have already pointed out in discussing the results of Sugar, Amador, and Griponissiotis (1950) there are few or no callosal fibres from either PB or PC in the macaque; Bailey, Bonin, and McCulloch (1950) found no callosal fibres from PC in the chimpanzee (they did not study PB). It is evident that callosal connexions of the somesthetic cortex must be by way of the somesthetic association cortex which was largely removed by Ettlinger and his colleagues in their experiments. The posterior parietal ablation is thus equivalent to a callosal ablation. This equivalence is based on the fact that the callosal fibres involved in transfer between the somesthetic association cortex of the two sides originate and terminate in the posterior parietal regions. It is highly likely that many of the effects of posterior parietal lesions in man depend similarly on the destruction of the origin and termination of callosal fibres. This will be discussed in a later section of the paper.

The third effect of unilateral posterior parietal ablations in Ettlinger and Kalsbeck's experiments was a disturbance of reaching with the contralateral hand. This disturbance was both visual and nonvisual. Nonvisual reaching was tested by having the monkey reach with one hand for a pellet held in the other restrained hand. This difficulty is readily explained on the basis that the posterior parietal ablation has destroyed the callosal connexions between the two somesthetic association areas. Hence it might be difficult for one hand to reach an object held in the other. This disturbance is comparable to the effects seen in the patient of Geschwind and Kaplan (1962b) to be discussed later in the paper who could not draw with one hand an object held in the other.

The disturbance of visual reaching is at first apparently susceptible to a similar explanation but certain difficulties arise which make it uncertain whether this explanation is in fact correct. There are at least two mechanisms by which we could conceive that visual reaching might take place. There are almost certainly no significant direct connexions from the visual to the motor cortex. Chusid, Sugar, and French (1948) have shown that there are important connexions in the macaque from the depths of the lunate sulcus (i.e. visual association area) to the arcuate sulcus (area 6), from which there are connexions to the motor cortex proper (Bonin and Bailey, 1947). This would thus constitute a pathway for carrying out

motor activities under visual control. Alternatively there is a path from the visual cortex to brain-stem motor mechanisms. These subcortical motor connexions do not arise from the visual cortex proper but rather from the parieto-occipital junction; this region thereby becomes a supplementary motor area (Crosby *et al.*, 1962). The pathway may then descend to the pons and after relay in the pontine nuclei reach the cerebellar hemisphere of the opposite side (Jansen and Brodal, 1954). Whether the cortico-cortical and cortico-subcortical pathways are equivalent or whether one might not subserve more precise reaching remains to be determined. A large unilateral parieto-preoccipital lesion such as that produced by Ettlinger and his colleagues should destroy both these visual-motor pathways in the involved hemisphere. One might therefore expect that following such a unilateral ablation there should be a defect of reaching by the hand contralateral to the ablation in the visual field contralateral to the ablation. By contrast reaching by the hand ipsilateral to the ablation in the visual field ipsilateral to the ablation should be normal. Both these effects were seen in the animals of Ettlinger and his co-workers.*

The problem becomes, however, more complex when we consider 'crossed' reaching, e.g. reaching by the right hand in the left visual field. That this type of activity depends on callosal connexions is made likely by the experiments of Downer (1959), in which the callosum and chiasm had been sectioned. When one eye was covered in these animals, control of the hand ipsilateral to the uncovered eye was very poor while control of the contralateral hand was normal.[7] It appears not unlikely from Downer's descriptions that his monkey showed a defect of visual reaching such as Ettlinger *et al.* described. There appear to be two reasonably possible pathways for this transcallosal visual reaching. Let us assume for simplicity that what is required is reaching with the left hand in the right visual field. One pathway would go from the left visual cortex to the left visual association area; from there it would proceed to the region of the arcuate sulcus (i.e. 'motor association area' or area 6) of the same side, then to the arcuate sulcus region of the opposite side and finally to the motor cortex of the opposite side. If this were the pathway the effect of a large parieto-preoccipital region should be to produce difficulties in visual reaching with *both* hands in the *right* visual field but with neither hand in the left. This pathway may well be the correct one for visual

reaching in man. The difficulty in reaching with either hand in one visual field is a sign of what some authors have called 'visual disorientation' in a half-field (Brain, 1941; Ettlinger and Kalsbeck, 1962). In the experiments by Ettlinger and his colleagues the animal made errors in reaching with the contralateral hand in either visual field, which is not in accord with this anatomical route.

An alternative pathway for crossed movements would be one that proceeded from the visual cortex to the visual association areas of one side, then crossed the callosum to the opposite visual association area and then ran forward to the motor association region in the vicinity of the arcuate sulcus, and finally reached the motor cortex. The effects of a parieto-preoccipital lesion might depend on exactly where the callosal fibres crossed. If they crossed posterior to such a lesion then the animal would show (1) a defect of visual reaching on the contralateral side with the contralateral hand, since the lesion would effect a visuo-motor disconnexion in the operated hemisphere; (2) a defect of visual reaching with the contralateral hand in the field ipsilateral to the ablation. This pathway would proceed via the right visual association area to left visual association area and then forward via the posterior parietal region to the motor region; (3) visual reaching with the ipsilateral hand would be preserved in both visual fields.

This pattern of defect and preservation outlined under the headings 1–3 is in fact what Ettlinger and Kalsbeck found. This pathway is therefore a possible one.

There is of course one further and very likely possibility. This is that both of the pathways listed above are actually used in the intact animal in carrying out crossed reaching and that disruption of either will lead to difficulties in crossed reaching which may, however, not be permanent since restitution may occur via the spared pathway. More stable difficulties should follow disruption of both pathways.

In any event the views expressed here are susceptible of further experimental test. One simple test of the theory would involve extending the unilateral lesions produced by Ettlinger and Kalsbeck a short distance posterior to the lunate sulcus. This should probably lead to a disruption of the callosal pathway between the right and left area 18; the animal should then show normal reaching only with the hand ipsilateral to the ablation in the ipsilateral field.

A third alternative is to assume that visual reaching in one field in the *monkey* depends on a subcortical pathway from visual association cortex which projects bilaterally, thus excluding callosal participation. This would not, however, be consistent with Ettlinger and Kalsbeck's results.

It is very likely that in man certain effects of parietal lesions are based on visual-motor disconnexions, a view which in part goes back to Liepmann (1900, 1906). We have mentioned this briefly above in regard to disturbances of visual reaching in man but will return to it more extensively in our discussion of the apraxias.

In summary I would hypothesize that posterior parietal ablations such as those described by Ettlinger produce a triple disconnexion (1) between somesthetic cortex and limbic structures, (2) between the somesthetic cortex on the two sides, (3) between visual and motor regions. Of these three mechanisms probably that listed under heading (3), i.e. the visuomotor disconnexion, presents the greatest difficulties of interpretation.

9. *The Auditory System*

I will not discuss the auditory system in detail. Its precise cortico-cortical connexions in primates are poorly known as a result of the poorly accessible location of the primary auditory cortex in the Sylvian fissure of these animals. As Bailey *et al.* (1950) point out, it was difficult, on the basis of their experiments, to separate the connexions of the primary auditory cortex (TC) from those of the para-auditory cortex TB in the chimpanzee since both of these lie on the supratemporal plane in the Sylvian fissure. The studies on the macaque suggested that TC fired TB, and TB fired TA (the posterior end of the superior temporal gyrus). In neither monkey nor chimpanzee did they find any callosal connexions between TA of one side and that of the other. We would expect if Flechsig's rule holds here that the callosal connexions which do in fact exist between the two auditory regions on the supratemporal plane would proceed from area TB on one side to the same area on the other side rather than between the two areas TC (the primary auditory cortex). This, however, remains to be precisely confirmed.

Stepien *et al.* (1960) removed the first and second temporal convolutions anterior to the primary auditory area bilaterally in African green monkeys. These monkeys failed on a task involving auditory recent memory. Their experiments are similar to those reported by Goldberg,

Diamond, and Neff (as summarized by Ades, 1959) who showed that removal of the so-called insular and temporal cortex of a cat (i.e. the cortex ventral to the para-auditory areas AII and EP) led to loss of simple tone discrimination and tonal pattern discrimination with subsequent failure to reacquire tonal pattern discrimination. Stepien *et al.* point out that the lesions they produced in monkeys were probably homologous to those produced by Goldberg *et al.* in cats. It should be pointed out, however, that such homologies may not be precise. As I have pointed out earlier for the visual system, there is some suggestion that the distinction between primary receptive areas and association cortex is less sharp in animals lower on the phylogenetic scale. It is therefore possible that the auditory receptive area and association cortex are also more sharply distinguished in the primates than they are in lower forms. Further research on the auditory system of primates should help to clarify these questions further. Important steps in overcoming the technical difficulties in this field were made by Pribram *et al.* (1954); further advances in knowledge may be expected to result from such improvements in technique.

In interpreting the effects of the removal of auditory association areas, Stepien *et al.* (1960) noted that removing the rhinencephalic portions of the temporal lobe led to recent memory defect in all modalities. They comment, "The mechanism whereby these rhinencephalic portions interact with neocortical functions... is unknown." They go on to quote from a recent paper by Jasper and Rasmussen: "... We may assume that separate projections of neocortical and rhinencephalic structures to common centrencephalic systems... are of critical importance.... On the other hand, rhinencephalic formations may act as an intermediate way station in the conduction of impulse patterns from neocortex to brain-stem or conversely for impulses arriving to the cortex from subcortical structures...." I would tend to stress the latter of these hypotheses and to state further that the association areas in turn act as way stations between primary receiving areas and rhinencephalic structures.

10. *The Problem of Mirror Foci*

The concept of disconnexion from the rhinencephalon may be useful in explaining certain aspects of the physiology of mirror foci. Morrell has speculated that the development of a mirror focus on the side opposite a

primary epileptogenic lesion may represent a 'learning' situation by the uninvolved side. He has shown (Morrell, 1960) that an area of cortex in the rabbit isolated from all connexions except the callosal ones can develop a *dependent* mirror focus but will not develop an independent epileptic focus. Morrell sees this result as showing the importance of subcortical connexions in the establishment of an independent focus. I would agree with Morrell that this result is highly suggestive that the establishment of an independent mirror focus may be a 'learning' activity. While he stressed the importance of thalamic connexions, an alternative hypothesis is that the significant factor in his experiments was the isolation of the cortical slab from the hippocampal region, i.e. that he had produced a disconnexion from the limbic system. I have already stressed the importance of such disconnexions in animal learning. The hypothesis that the development of mirror foci at a particular site is a learning process dependent on the intactness of connexions from that site to limbic system leads to some interesting possibilities for research. The study of sites of development of secondary epileptic foci in higher animals may be a useful adjunct in tracing association systems.

It should be noted that theoretically there is no reason why independent secondary foci should not develop as readily within the same hemisphere as the primary focus as within the opposite hemisphere. Furthermore mirror foci should be rare when commissural connexions are lacking, as is true of the classical receptive and motor areas.

11. *Disconnexions from the Limbic System in Man*

This paper has so far considered disturbances of the higher functions in animals and has placed emphasis on lesions disconnecting primary receptive centres from rhinencephalic structures. In a later section I will discuss disturbances of the higher functions in man and will present the thesis that those which have been observed generally do *not* depend on disconnexions from the rhinencephalon but on other mechanisms not extensively present in subhuman forms. The question at issue in this section is therefore a much more limited one: Do there exist in man disconnexions from the limbic system and if so, what are the manifestations of these lesions? There is certainly very little good evidence to aid in answering these questions and this section must be regarded as highly speculative.

It might be argued that syndromes of disconnexion from the limbic system homologous to those seen in monkeys have rarely, if ever, been seen clinically because the appropriate lesions are highly unlikely ones. I suspect, however, that an even more important reason is that the types of sensory-rhinencephalic linkages seen in subhuman primates are less common in man, their role being taken to a great extent by more indirect associations by way of the speech areas. This newer type of indirect association will be discussed in greater detail in the next section.

Lesions in man comparable to those in the lateral and basal temporal lobes in monkeys might thus not produce the same effects. For although the phylogenetically less advanced visual-limbic pathway was destroyed, there would still remain a pathway going from the visual association cortex (probably via the angular gyrus) to the temporal speech area, i.e. part of the auditory association cortex and from there proceeding to the limbic system. This type of indirect pathway might be present in a monkey but as I have already commented only in much attenuated form.

There are, however, some clinical conditions which arouse speculations as to whether they might not be the result of disconnexions of cortical regions from the limbic system. One of these is the syndrome first described by Schilder and Stengel and called by them 'asymbolia for pain' (Rubins and Friedman, 1948). In this condition the patient correctly distinguishes sharp from dull but shows no response to pain or even to threatening gestures. Some of the patients explicitly denied feeling pain. On the other hand, it was quite characteristic of these patients to respond appropriately to verbal threats. Schilder and Stengel (1928) pointed out that their original case also showed an indifference to loud noises.

A striking feature of this condition is the fact that it tends to be strongly associated with lesions of the dominant parietal lobe. Schilder and Stengel (1931) brought post-mortem evidence that the supramarginal gyrus was the structure involved. The clinical picture of Rubins and Friedman's cases was strongly compatible with the diagnosis of left parietal disease (e.g. the various components of Gerstmann's syndrome were frequently present).

In order to put these data together – speculatively – let us further note the fact that some observers have suggested that the cortical end-stage of the pain pathway lies in the supramarginal gyrus. This would correspond with the second sensory area (*see* discussion by Sweet, 1959). Biemond

(1956) found evidence for this localization in three cases. Let us assume now that a patient develops a lesion not of the secondary sensory area but of the connexions between it and the limbic system. It is conceivable that while the patient could still distinguish the qualities of the stimulus, he would have no emotional response to it. This distinction between pain as a sensation and the emotional response to pain has long figured in the literature of research on pain. Denny-Brown (1962) has stated this distinction well, "...for such patients (i.e. those with asymbolia for pain) that we have seen can feel pain and can discuss it, though it is not of any biological importance to them." What I am suggesting here is that there may be an anatomical basis for this distinction.

My speculation would be that the connexions from the secondary sensory area to the limbic system would go by way of insular cortex. The lesion causing pain asymboly would in fact spare the secondary sensory area but involve perhaps parietal operculum and insula, cutting off the connexions to the limbic system. Schilder and Stengel's first case showed impairment of response to unpleasantly loud noises. It is possible that the secondary auditory area near the secondary somatic area may similarly be part of the pathway from auditory system to limbic system which was also cut off by the lesion. These explanations would also be consistent with the fact that these patients may respond to verbally expressed threats which presumably can still reach the limbic system by connexions from the posterior speech area on the lateral surface of the temporal lobe.

This mechanism might be approached profitably via animal experimentation. Conceivably it might even have practical usefulness since a reasonably discrete lesion might be available for the patient with intractable pain. As Sweet (1959) points out, the syndrome of asymboly for pain is quite unlike the syndrome resulting from lobotomy in which the patient's physical reactions to pain are, if anything, increased and who verbally readily admits to pain. The effectiveness of lobotomy is probably based on some other mechanism, perhaps decreased attentiveness to a chronic pain. Conceivably pain asymboly could be achieved without the serious emotional and judgmental effects of many lobotomies.

The same mechanism might possibly play a role in the syndrome of congenital indifference to pain which bears a definite resemblance to asymboly for pain. Patients with this congenital disturbance also appear to show excellent appreciation of painful stimuli as sensations but demon-

strate little emotional arousal by such stimuli. Furthermore these patients differ from normals in another striking way. Normals appear to learn readily that certain stimuli will lead to pain and consequently can learn to develop almost automatic avoidance movements to such stimuli (e.g. the rapid withdrawal movements made on touching an unexpectedly warm kitchen utensil). Patients with congenital indifference seem to fail to acquire these and consequently often suffer considerable tissue damage. It is not unreasonable to speculate that the combination of inadequate emotional response to painful stimuli and the inability to learn appropriate responses to such stimuli may both perhaps be the result of failure to form the normal connexions between the cortical regions involved in pain perception and the limbic system. Whether the congenital lesion would involve a bilateral failure of development of those regions involved in acquired asymboly for pain remains of course to be determined.

I would like to stress again the speculative character of this discussion and the evident problems it raises. Why should asymboly result from a *unilateral* parietal lesion? Why do the patients show a lack of response to *visual* threat? Even more basic objections might be raised against the above discussion. On the one hand, the thesis that the cortical representation for pain is in the secondary somatic area is certainly not conclusively proven. On the other hand, certain authors such as Weinstein *et al.* (1955) have cast doubt on the occurrence of pain asymboly as a localizable phenomenon. Sweet (1959) appears to be sympathetic to their view. The arguments of these authors, however, while cogent, are not conclusive. The whole problem of asymboly and its possible anatomical substrates needs new evaluation.

Another possibility to consider is that disconnexion from the limbic system may play a role in certain situations in man in which there is denial of illness. It has often been observed by clinicians that there is a marked difference in the emotional reaction to their illness of patients with aphasias resulting from frontal lesions and of those with temporal lesions. The frontal group are typically depressed and acutely aware of their disabilities. The patients with temporal lesions are frequently euphoric, often behave as if they are unaware of their aphasia and may even actively deny any disability. One possibility to account for the behaviour of the temporal group is that the temporal lesion may in damaging Wernicke's area also lead frequently to the cutting off of connex-

ions to the limbic system from this part of the temporal lobe. This lesion might therefore lead to a failure of the disability to arouse emotional responses. Another effect of such a disconnexion of the speech area from the limbic structures might be to cause difficulties in verbal learning[8] and thus make rehabilitation more difficult. I would like to stress that the mechanism here speculatively raised as a cause of denial of illness is certainly not the only one. I will discuss other possible mechanisms of denial later. I leave unsettled the issue as to whether the syndrome discussed in this section or any other syndromes in man are the result of disconnexions from the limbic system.

III. DISCONNEXION SYNDROMES IN MAN

1. *The Anatomical Basis of Language*

Man was the first species in whom disconnexion syndromes were clearly delineated. The writings of Dejerine and Liepmann mentioned in the introduction constitute the great landmarks of the early period of this type of investigation. I have placed the human material later in this paper because I feel it makes more sense to study it from the point of view of the evolution of the nervous system. As I have pointed out earlier, many of the discussions presented in this section can, in fact, be considered independently of the evolutionary hypotheses; despite this fact, I believe that these hypotheses may aid in bringing order into the material and in stimulating the design of specific experiments.

The preceding parts of this paper have cited the evidence that in lower mammals, the primary projection areas of the cortex subserve certain functions which tend subsequently to be separated in the primates. In keeping with this relatively minor degree of separation of functions, only a few regions of differing cytoarchitectonic structure are distinguishable. As we ascend the phylogenetic scale, the associative activities become separated to a great extent from the receptive. Large association areas more clearly separable from primary projection areas appear, and cytoarchitectonic differentiations increase. In accordance with the principle of Flechsig (which is applicable to man and the other primates but not to subprimate forms), the primary projection areas now send their connexions primarily to the immediately adjacent association cortex (parakoniocortex); the long connexions (either within a hemisphere or between

hemispheres) between different cortical regions take place predominantly between parts of the association cortex. To a great extent the most important connexions of the association cortex are with the neocortex of the temporal lobe (and perhaps also of the insula) which in turn feeds into limbic structures. In keeping with this, connexions involving linkages between any one sensory modality and the limbic system tend to be powerful (these connexions subserve emotional and autonomic responses to sensory stimuli, associations between one sensory modality and gustatory or olfactory stimuli, etc.) while other non-limbic sensory-sensory connexions tend to be weak. I have, in the first part of this paper, discussed in detail the effects of lesions separating the primary sensory modalities from the limbic structures in the primate.

The situation in man is not simply a slightly more complex version of the situation present in the higher primates but depends on the introduction of a new anatomical structure, the human inferior parietal lobule, which includes the angular and supramarginal gyri, to a rough approximation areas 39 and 40 of Brodmann. In keeping with the views of many anatomists Crosby *et al.* (1962) comment that these areas have not been recognized in the macaque. Critchley (1953), in his review of the anatomy of this region, says that even in the higher apes these areas are present only in rudimentary form. In keeping with the late evolutionary development of this region are certain other findings. The gyral structure of this area tends to be highly variable. In addition this area is one of the late myelinating regions or 'terminal zones' as Flechsig termed them. In fact, this region was, in Flechsig's map, one of the last three to myelinate. DeCrinis (cited by Bonin and Bailey, 1961) showed that part of this region is one of the last cortical areas in which dendrites appear. Yakovlev (personal communication) has pointed out that this region matures cytoarchitectonically very late, often in late childhood. In addition, he has pointed out that preliminary studies suggest that this region receives very few thalamic afferents. In this respect it is similar to part of the frontal association area which is also largely athalamic; this part of the frontal lobe is also phylogenetically new, myelinates late and forms dendrites late. The afferent connexions of this new parietal association area may therefore be predominantly from other cortical regions. As an association area, this region is also different from the older association areas in not being essentially concentric with one of the primary projection centres.

The newness of this region is also reflected in another anatomical feature probably unique to the human brain. G. Elliot Smith (1907) studied distinctions of cortical architecture based on naked-eye appearances of the freshly cut brain. He found that his inferior parietal area A (roughly corresponding to the region I have been discussing here) was bounded above and below by thin distinctive bands of cortex. The lower is the so-called 'visuo-auditory band'. As Elliot Smith comments, "This attenuated band is all that is left of the extensive bond of union between these two areas which in the lower mammals have co-extensive borders: in man and to a less extent in the apes the great development of the inferior parietal area above it and the temporal areas below it have pushed these two parts asunder, leaving this narrow connecting bridge. In support of this hypothesis of the primitive nature of the band, I might call attention to the fact (which Flechsig has clearly established) of its early medullation...." The upper band is the 'visuo-sensory band', another thin band of cortex running along the superior lip of the intraparietal sulcus. Flechsig had shown that this strand also undergoes early myelination. Elliot Smith comments, "It is the attenuated fragment of that extensive connexion between the visual and sensory areas of the brain which has remained after these areas have been pushed apart by the great expansion of the parietal areas...." Cytoarchitectural studies such as those of von Economo and Koskinas have confirmed the existence in this band of cortex of structure different from that of the cortex above and below the band.

Some authors (e.g. Konorski recently) have interpreted certain clinical syndromes as disconnexions between visual and other sensory spheres resulting from lesions of these bands. I believe, in fact, that the primitive character of the bands and their small size make these interpretations unlikely; more probable is that the observed phenomena resulted from lesions of the adjacent portions of the inferior parietal lobule.

We thus have this extensive, evolutionarily advanced, parietal association area developing not in apposition to the primary projection areas for vision, somesthetic sensibility, and hearing but rather at the point of junction of these areas as Critchley (1953) has indicated. This region possibly being one of few thalamic connexions may well receive most of its afferents from the adjacent association areas; it is thus an association area of association areas. In more classical terms, it would be called a secondary association area. The probable significance of this anatomical location is

heightened by reference to our earlier discussion of subhuman forms. In these it appears as if association areas feed into temporal neocortex relaying in turn to limbic and rhinencephalic structures. As I pointed out in the earlier discussion, cross-connexions between primary nonlimbic sensory modalities are weak in subhuman forms. In man, with the introduction of the angular gyrus region, intermodal associations become powerful. In a sense the parietal association area frees man to some extent from the limbic system. This independence is only relative since ultimately learning still depends, even in man, on intact connections with limbic structures. The well-known permanent severe disturbance of new learning resulting from bilateral lesions of the hippocampal region attests to this fact (Scoville and Milner, 1957).

The development of language is probably heavily dependent on the emergence of the parietal association area since at least in what is perhaps its simplest aspect (object naming) language depends on associations between other modalities and audition. Early language experience, at least, most likely depends heavily on the forming of somesthetic-auditory and visual-auditory associations, as well as auditory-auditory associations. Whether this great association area is as powerfully involved in mediating other cross-modal associations (e.g. visual-tactile) is not clear. Situations which demand these other types of cross-modal association appear to be less important than those involving audition, probably because language depends on this latter type of association. Perhaps in the deaf person learning written language tactile-visual associations become important. Critchley (1953) comments that it is tempting to associate the growth of the postparietal region with the development of speech. I would think that the parietal region is involved in the development of speech because of its importance in enhancing cross-modal associations. As I have noted earlier, *it cannot be argued that the ability to form cross-modal associations depends on already having speech;* rather we must say that *the ability to acquire speech has as a prerequisite the ability to form cross-modal associations.* An important area of research which remains to be studied extensively is that of the course of acquisition of cross-modal learning in childhood before speech is fully developed.

The objection might be raised that in some congenitally deaf people language is learned entirely in the form of visual-visual associations. If we restate the principle stated above in somewhat more precise form it will

be seen that this objection is readily met. *In sub-human forms the only readily established sensory-sensory associations are those between a non-limbic (i.e. visual, tactile or auditory) stimulus and a limbic stimulus. It is only in man that associations between two non-limbic stimuli are readily formed and it is this ability which underlies the learning of names of objects* (Geschwind, 1964b).

It is also not unlikely that the development of cerebral dominance is related to greater development of this new parietal association area. Bonin (1962) has discussed this problem and stressed the smallness of the differences between hemispheres. However, the results which he himself quotes as well as those cited by Connolly (1950) do, in fact, tend to support the view that the left hemisphere is the more developed, at least as far as fissural pattern is concerned, and it is quite possible that Bonin's assessment of the data is much too conservative. I would speculate that left cerebral dominance is based on (or indeed perhaps equivalent to) the ability of the left hemisphere more readily to make cross-modal associations, an ability perhaps based on greater development of the left posterior parietal region. A detailed discussion of dominance would, however, lead us too afar afield.

We will simply assume from here on that the left hemisphere is dominant for speech functions and that this dominance depends on enhanced activity of the left speech area. The most important part of this area is the middle and posterior portions of the superior temporal gyrus which are, of course, part of the auditory association area and form the classical Wernicke's area. Connexions from other sensory modalities, at least vision and somesthetic sensation, are assumed to come to this speech zone by way of the angular gyrus region. Connexions from the speech area to other sensory parts of brain (i.e. connexions which subserve the arousing of tactile and visual associations by auditory stimuli in general and speech in particular) are presumed to go in the reverse direction by way of the angular gyrus region. An important area of research is suggested by these briefly stated assumptions: the detailed pattern of connexions between the angular gyrus and the specified regions of the superior temporal gyrus (roughly area 22 of Brodmann) deserves careful elucidation.

I would like to point out here that although the predominance of the human parietal association areas is generally admitted not all authors would give them as much prominence as I have. Thus Bonin and Bailey

(1947) state, "We cannot agree... that the homologues of Brodmann's areas 39 and 40 in man exist in the macaque only as very small patches..." These same authors, however (Bonin and Bailey, 1961), stress that the part of the brain which increases in man most strikingly is not the frontal lobe but "the parietal and temporal lobe in the widest meaning of that term, and it is here that we should look for the substrate of certain functions which are supposed to be characteristic of man." They quote with approval Weidenreich's statement that the growth of the brain in man affects primarily the parietal lobes and the posterior region of the inferior part of the temporal lobe. At any rate, if these authors deny the marked parietal predominance that I have stressed they at least admit a relative predominance of this region in man. The exact degree of the uniqueness of the inferior parietal region in man remains to be determined.

In the preceding paragraphs I have outlined some of the new elements that must be considered in evaluating disturbances of the higher functions in man. In animals I have stressed disconnexions from the limbic system. In man with the development of speech, Wernicke's area becomes of major importance. Disconnexion syndromes will result from lesions which cut off Wernicke's area from primary sensory areas. Some of these lesions will lie in the white matter of the hemispheres while others will involve the cortex of the angular gyrus which probably acts as a way station between the primary sensory modalities and the speech area. In addition lesions which cut off connexions from Wernicke's area to motor portions of the hemispheres will lead to profound effects on behaviour.

In the following sections I will specify in greater depth some of the clinical and anatomical evidence which supports the model I have sketched. I will first consider lesions which lead to modality-specific disturbances by isolating specific sensory projection regions from the speech area. The lesions producing these disconnecting effects may be either in white matter systems such as the corpus callosum or in the association cortex giving rise to these fibre tracts. This discussion of highly specific receptive aphasic disturbances will lead us into a discussion of a related group of impairments, the agnosias. Similarly, I will consider disconnexions of this posterior temporal speech area from the motor systems, which will lead us into a discussion of the apraxias. Finally, I will consider disconnexions of the posterior speech area from the anterior (frontal) speech region.

2. *Pure Word-blindness Without Agraphia*

This condition must be regarded as of special importance since it is probably the first example of a callosal disconnexion syndrome for which clear anatomical evidence was forthcoming. I have discussed Dejerine's (1891, 1892) classic papers elsewhere in detail (Geschwind, 1962) and will only summarize here. Dejerine developed his analysis of word-blindness on the basis of the findings of two patients reported in consecutive years. The first patient (Dejerine, 1891) showed the clinical picture of pure alexia *with* agraphia in the absence of other significant aphasic disturbances. The second patient had by contrast the syndrome of pure alexia *without* agraphia. The information from the two cases combines to form a simple picture of the mechanisms of disturbances of reading. Before discussing pure word-blindness without agraphia, I will present first the findings in alexia with agraphia.

The first paper (Dejerine, 1891) described a 63-year-old man who developed the sudden onset of inability to read and write in the absence of other significant neurological disabilities except for a right hemianopia. At post-mortem (eight months after the onset) the brain was entirely normal except for a lesion involving the inferior three-quarters of the angular gyrus and penetrating inwards to the occipital horn of the lateral ventricle. The inward extent of the lesion had, of course, involved the optic radiations. Dejerine concluded that the lesion had destroyed a 'visual memory centre for words' with resultant loss of the ability to comprehend written language or to write. Within a year Berkhan and Serieux (cited by Dejerine, 1892) had published similar cases with similar localization.

The second paper (Dejerine, 1892), longer and more detailed than the first, describes a patient followed by Dejerine over a period of more than four years. This patient suffered from the acute loss of the ability to read letters, words or musical notation in association with a right hemianopia. He could copy words correctly but could not transcribe print into script; he could write correctly (in script) either spontaneously or to dictation but could not read what he had written a short time previously. Although he could not read 'visually', he could 'read' by tracing the outlines of letters with his hand and could recognize the letters formed by having the examiner move his hand passively through the air. Although he could

not read, he was able to name even extremely complex objects such as pictures of scientific instruments in a catalogue. There was no evidence of any general intellectual disturbance since the patient continued during his illness to operate a highly successful business, to gamble at cards successfully, and to learn vocal and instrumental parts of operas by ear since he could no longer read music. Ten days before death he suddenly developed an agraphia. At post-mortem the brain showed an infarct of the left occipital lobe and of the splenium of the corpus callosum. The occipital infarct was shrunken and yellow and adherent to the overlying meninges, all of which indicated a lesion of considerable age. By contrast the patient showed a fresh infarct of the left angular gyrus which must have led to the new symptomatology ten days before death.

Dejerine interpreted this case as a disconnexion of the visual cortex from the speech area. Since the left occipital cortex was destroyed, this patient could perceive words only in the left visual field, i.e. only in the right occipital cortex. It is, however, not possible to read with the right hemisphere alone since destruction of the left hemisphere produces an alexia as one part of a gross aphasic syndrome. The visual stimuli received in the right visual cortex must therefore be transmitted to some region of the left hemisphere. It would seem reasonable on the basis of the findings of the first case discussed to assume that the relevant region in the left hemisphere is in the angular gyrus. The extensive lesion of the white matter of the left occipital lobe and of the splenium of the corpus callosum, however, cut off the connexions between the right occipital lobe and the left angular gyrus. Dejerine therefore argued that pure word-blindness without agraphia resulted from disconnection of the intact right visual cortex from the left angular gyrus in a patient in whom the left visual cortex had been destroyed.

The preservation of the left angular gyrus explains several aspects of the syndrome of alexia without agraphia. Thus, the preserved ability to write suggested to Dejerine that the 'visual word-centre' was intact. The ability to 'read' tactilely clearly relies on the fact that the pathway to the angular gyrus via the somesthetic system is intact.

There is one further difference between pure alexia with agraphia and pure alexia without agraphia which supports the Dejerine interpretation of the former syndrome as the result of a lesion of a 'memory centre' and of the latter as a disconnexion from this 'memory centre'. Dr. Davis

Howes and I have had the opportunity to observe the spelling performance of a patient with pure alexia with agraphia. This patient had normal spontaneous speech. He was unable, however, to spell correctly even the simplest word. Similarly, although he understood complex spoken sentences, he could not understand even three- or four-letter words when they were spelled to him. By contrast the two patients with pure alexia without agraphia whom I have observed (one in collaboration with Dr. Howes and one with Dr. Michael Fusillo) have been able both to spell and to comprehend simple spelled words. The explanation of this phenomenon derives from the fact that spelling is learned only as part of learning to read and write. In order to comprehend a word spelled out loud, the listener must transform it into written form and then 'read' it. Conversely, to spell orally one must transform the spoken word into its written form and then 'read' the letters one by one. One can state this argument more simply by noting that a loss of visual word-memory returns the patient to the state of being illiterate; lack of reading, writing, and spelling and incomprehension of spelled words are all components of this more primitive state.[9] The patient with pure alexia without agraphia preserves the ability to spell since he still preserves the 'centre' which turns spoken into written language and also carries on the reverse operation.

Parenthetically it should be noted that this disturbance of spelling gives us a particularly useful clue as to the function of the part of the angular gyrus involved in 'visual word memory'. It is a region which turns written language into spoken language and vice versa. It is, in short, a region specifically designed for carrying on visual-auditory cross-modal associations in both directions and indeed for storing the memory of the 'rules of translation' from written to spoken language. I will return to this point later on.

It should be pointed out that Dejerine's paper described only the gross findings in the brain of the patient with pure alexia without agraphia. Vialet (1893) published a year later the detailed description of the central nervous system which had been cut in whole brain sections.[10] The lesion described by Dejerine for pure alexia without agraphia was soon confirmed by other authors. Bastian (1898) only a few years after Dejerine's publication was able to cite several cases where the lesion had involved the left occipital cortex and the splenium of the corpus callosum.

Many facts can be marshalled to show the importance of the lesion of the splenium which acts to disconnect the right visual region from the angular gyrus. Foix and Hillemand (1925) pointed out that one patient who at post-mortem had an infarct of the left visual cortex without involvement of the splenium had had no alexia in life; another patient with an infarct of the left visual cortex and in addition destruction of the splenium had shown the syndrome of alexia without agraphia. As I have pointed out elsewhere (Geschwind, 1962), the lack of this syndrome after penetrating head trauma results from the fact that a missile is very unlikely to destroy the left visual cortex and the splenium of the corpus callosum. The study of Hécaen *et al.* (1952) showed that alexia invariably occurred after left occipital lobectomy but was transient in all cases, clearing in a few months. The splenium, of course, was left intact so that there was a path from the right occipital cortex to the left angular gyrus. The case of Trescher and Ford (1937) and the cases of Maspes (1948) who had the splenium cut in the course of removal of a colloid cyst of the third ventricle all developed alexia in the left visual field. By contrast, the patient of Geschwind and Kaplan (1962b) in whom there was no alexia of the left visual field showed at post-mortem an intact splenium although the anterior four-fifths of the callosum was infarcted. The patient of Gazzaniga, Bogen and Sperry (1962) in whom the splenium was cut showed an alexia in the left visual field.

By contrast to the above results, Akelaitis (1941b, 1943, 1944) described six patients in whom the splenium had been cut and who showed no alexia in the left visual field. I will defer a critique of these discrepancies to a later section of the paper where all the Akelaitis results will be discussed.

A further anatomical point deserves discussion. The first is the exact path of the connexions between the right visual cortex and the angular gyrus. Since the visual cortex has no callosal fibres, this pathway must be by way of the association areas, i.e. the pathway goes from the right area 17 to the right-sided area 18 (Myers, 1962a) and from this it eventually crosses the callosum.

I can conceive of three possibilities for the course of this pathway: (1) The pathway proceeds from the right area 17 to the right visual association areas, from there to the right angular gyrus and finally across the corpus callosum to the left angular gyrus. (2) The pathway runs from the right area 17 to the right visual association areas, then crosses the callosum

to the left visual association areas and finally runs forward to the left angular gyrus. (3) The third possibility is that both pathways are used. This possibility is the one that would appear most likely under the assumption that we are dealing with an equipotential system in which a part can take over some of the functions of the whole.

Possibility 2 is ruled out as the *exclusive* pathway by the fact that no permanent alexia results from left occipital lobectomies (Hécaen *et al.*, 1952). But that there is some participation of this pathway is made highly likely by the fact that the alexia from left occipital lobectomy does last for several months, too long for the effect to be due to post-operative oedema but long enough for pathway 1 to come to take over the role completely. There is probably some permanent effect of destroying pathway 2 since as Hécaen *et al.* (1952) point out, their patients with left occipital lobectomies disliked reading even after their ability to read had returned.

It is likely that pathway 1 also participates normally since patients with right parietal lesions may show a failure to read the left halves of words despite an intact left visual field (Kinsbourne and Warrington, 1962). The localization of the lesions in this latter paper, however, is not certain and more studies will be needed. The conclusions are, however, in keeping with the clinical observations of others on alexias from right parietal lesions.[11] It would thus appear that both pathways are normally used, i.e. possibility 3 is the correct one.

If we refer back to our earlier discussion of the possible functions of the angular gyrus, we can speculate as to the mechanism of its function as a visual memory centre for words. The angular gyrus, as we have noted already, becomes a memory for written words by acting as an area for forming – and storing – cross-modal associations between vision and hearing. It seems likely that this store of cross-modal associations involves more than words. An analysis of what is lost and preserved in pure alexia without agraphia may help to clarify this point.

While the reading aloud and comprehension of written words is lost, the ability to name and recognize objects is preserved. We can expand a suggestion by Adolf Meyer (1905) to develop the explanation for this. Objects have rich associations in other modalities, e.g. we can recognize an apple by vision, touch, taste, smell, even by its texture on being bitten. The arousal of such associations permits the finding of an alternative pathway across an uninvolved more anterior portion of the corpus

callosum. The reading of numbers is also frequently preserved in these cases – in Dejerine's case number reading was perfect. Other authors, e.g. Symonds (1953), have discussed this striking fact. The learning of numbers is also associated with heavy somesthetic reinforcement (counting on the fingers) which frequently persists for a long time in childhood because the child can use his own fingers for this purpose. By contrast, reading is learned, except in the very earliest stages, as a pure visual-auditory task.

A difficulty with colours is common in these cases. Dr. Michael Fusillo and I (Geschwind and Fusillo, 1964) have recently studied a case of pure word-blindness with persistent difficulty in colours. We were able to show that this was a pure difficulty in colour-*naming*. Thus the patient matched colours by hue without error despite large differences in brightness and saturation. He would without error identify the figures on two different pseudo-isochromatic tests of colour vision. It is obvious that a colour has no smell, taste, or feel – the only association unique to the colour is its name. The loss of colour-naming is thus another example of loss of visual-auditory associations. The loss of ability to read music, as in Dejerine's case, appears to be another example of loss of visual-auditory associations.

I would like to stress the fact that many combinations of lesions may lead to the same syndrome. I recently observed a patient who had suffered a cerebral vascular accident which seemed likely to have been in the left posterior parietal region. A year later he developed a *left* hemianopia and became alexic. I wondered whether his initial lesion had not destroyed the connexions between his *left* visual cortex and his left angular gyrus so that he was reading only with the right occipital cortex until this was destroyed by a subsequent infarct. Unfortunately a post-mortem was not obtained and the above must remain pure speculation. This case illustrates, however, that it is a serious error to reject a case with multiple lesions since some interesting syndromes may result in such situations which could not be the effect of any single lesion.

Another area of speculation is the applicability of these results to failures of acquisition of reading, so-called congenital dyslexia. One possibility is that this syndrome is due to delayed development of the angular gyrus region – probably bilaterally. The results cited earlier that the angular gyrus region typically matures late make it plausible that a significant group will not have achieved adequate development by the time of the usual age of learning to read. The tendency for this condition to

disappear in many children with increasing age is compatible with the notion of slow maturation. The smaller proportion of girls showing this disturbance might be related to a more rapid maturation of the angular gyrus region in girls; this would be consistent with the more rapid attainment of most developmental milestones by girls. Study of an adequate number of anatomical specimens should make possible the verification or rejection of this developmental sex difference.

If the hypothesis of slow maturation is correct and if my views as to the possible functions of the angular gyrus region are correct, then certain predictions are possible. The child with congenital dyslexia should also show slower acquisition of colour-naming and music-reading. Reading of numbers should be more rapidly acquired. In fact, tests specifically designed to study cross-modal associations, particularly visual-auditory but also in the other modalities[12] might well be very rewarding. Birch (1962) has actually done preliminary studies on intersensory transfers in children and in particular in dyslexics. It will be most interesting to follow these pioneering studies.

It is probably necessary to study children as early as possible before language development has progressed very far and certainly before the learning of written language. I believe it would be possible to select a group in whom it could be predicted that the development of reading would be delayed on the basis of failures in learning other visual-auditory associations; it is conceivable that even the age of attainment of colour-naming might be a significant clue to the age at which reading can be acquired. Even casual observation among children shows a great variation in the age of acquisition of colour-naming among children in whom non-verbal testing shows colour-perception to be normal.

3. *Pure Word-Deafness*

Pure word-deafness probably has a similar pathogenesis to that of pure word-blindness without agraphia. Liepmann (1898) in a very carefully studied patient in whom ordinary deafness was clearly excluded showed that this syndrome could be produced by a unilateral lesion. The pathology was described in fuller detail by Liepmann and Storch (1902). The lesion, located subcortically in the left temporal lobe, had destroyed the left auditory radiation as well as the callosal fibres from the opposite auditory region. The lesion therefore had the effect of preventing the speech area

(i.e. that part of the auditory association cortex generally called Wernicke's area, which comprises the posterior portion of area 22 and occupies the posterior part of the superior temporal gyrus) from receiving auditory stimulation. The right primary auditory cortex could receive auditory stimuli but could not convey them to the speech area because the callosal connexions from the right side were destroyed in the left temporal lobe. This syndrome is rarer than pure word-blindness without agraphia for the obvious reason that a lesion which involves these structures usually extends into Wernicke's area and produces a more extensive aphasic picture. Some variation in the extent of the lesion causing pure word-blindness without agraphia would not lead to such obscuring symptoms.

The exact anatomy of the auditory cortex and of the callosal pathways between the two auditory regions is still uncertain in primates and man, in contrast to the more advanced state of knowledge of the anatomical arrangements in the cat (Ades, 1959). The primate data are less complete not only because of the smaller number of experiments but also because of the concealment of areas 41 and 42 in the supratemporal plane, i.e. within the depths of the Sylvian fissure. The crowding of structures in the supratemporal plane makes it particularly difficult to study the responses of TB (area 42) which is interspersed between the primary auditory cortex TC (area 41) and the rather extensive and on the whole readily accessible TA (area 22) on the lateral surface (occupying the first temporal gyrus in its middle and posterior regions). The cat data cannot be applied to the primate with confidence, not only because the anatomical homologies are not obvious but also because the danger would always exist that the distinction of primary receptive and association areas was more sharply defined in the phylogenetically advanced primates. We have already remarked that such a discrepancy between primate and feline anatomy exists in the visual system; while the cat's visual cortex according to some authors gives rise to callosal fibres that of the primate does not (Curtis, 1940; McCulloch and Garol, 1941; Bailey *et al.*, 1950; Myers, 1962a; Krieg, 1963).

The difficulties resulting from anatomical crowding on the supratemporal plane are reflected in the studies on the macaque where a clear-cut correlation between electrical response and cytoarchitecture has not so far been possible. The chimpanzee would probably represent a more suitable subject for this study because of the larger size of the brain. Bailey *et al.* (1943a) found in both the monkey and chimpanzee that auditory stimuli caused a large response in area 41 (TC) followed by a small one in area 42 (TB).

However, in a later publication on the chimpanzee Bailey, Bonin and McCulloch (1950) note, "It is impossible on the basis of our scanty data to separate surely the connexions of the auditory cortex (TC) from those of the para-auditory (TB). The efferent fibres seem to come mainly from the periphery, therefore, probably from TB." Sugar *et al.* (1948) studied the supratemporal plane in monkeys; they simply divided this region into five strips without regard to cytological differentiations between areas 41 and 42. They found the area of primary auditory response in the posterior third of the supratemporal plane. On strychninization this region fired the remainder of the supratemporal plane and also areas 22, 21 and 37; however, one cannot conclude with certainty that the primary auditory cortex itself fires these regions since the possibility must exist that the stimulated area may also have included part of area 42. These authors like McCulloch and Garol (1941) found a paucity of callosal fibres arising from area 22 or reaching area 22 from any part of the auditory system of the opposite side. Callosal fibres from one supratemporal plane to the other were plentiful but no distinction was made as to whether they arose from area 41 or 42. The data of Sugar and his co-workers suggest that there are more callosal fibres from the anterior portion of the supratemporal plane. This may correspond to the region in which Bailey *et al.* (1943a) saw small secondary responses and which they regarded as the anterior part of TB.

The suggestion that the main associative outflow of the auditory cortex is in the anterior part of the supratemporal plane receives some support in the work of Pribram *et al.* (1954). They found in the macaque that the region of short-latency responses to click lay posteriorly in the supratemporal plane; there was an anterior strip in which responses of much longer latency were seen. It would seem likely that these anterior regions are 'secondary' areas which are fired by the primary areas.[13]

We have presented these data in some detail to emphasize the tenuous nature of our knowledge of auditory association areas in the primate, and, obviously, in man. We might summarize roughly by saying that there appears to be general agreement that the centre of the primary auditory cortex lies in the posterior part of the supratemporal plane. Area 22 on the lateral surface of the first temporal gyrus constitutes a large area of auditory association cortex but is probably not the source of the callosal fibres of the auditory system. Callosal fibres probably arise from the supratemporal plane somewhat anterior to the primary auditory cortex. However, more detailed physiological study is needed to confirm even this rough picture. In addition a more careful study of the correlation of the pattern of transmission of impulses with cytoarchitectural differentiations is badly needed.

Clinical data perhaps may aid us in thinking about this problem and in suggesting further experiments in primates. As we noted at the beginning of this section Liepmann (1898) first described pure word-deafness from a *unilateral lesion*. There are, however, many more cases recorded of this syndrome from *bilateral* lesions. In these cases the most

common pattern has been that of bilateral often rather symmetrical cortico-subcortical lesions in the *anterior* Part of T_1, with Heschl's gyri intact. The subcortical penetration, particularly on the dominant side, is not very profound. These are the findings of Hoff (1961) but they generally coincide with those of other authors. Kurt Goldstein (1927) in his discussion of the localization of pure word-deafness places the lesion in the bilateral cases in the middle portion of T_1. I suspect that this is not a difference from Hoff's data since they were probably both emphasizing as the centre of the involved zone roughly the junction of the anterior and middle thirds of T_1. This zone is at the junction of area 42 with the anterior part of area 22. The precise mechanism of this lesion is not clear. One possibility is that the outflow from auditory cortex proper (area 41) goes to area 42, that the outflow path then continues from the region of junction of areas 42 and 22 posteriorly in area 22. The left-sided lesion would cut off the left auditory cortex from the left area 22; the right-sided lesion would cut off the origin of the callosal fibres (presumably coming from area 42) from the right auditory region. This interpretation would be in keeping with the findings in primates that area 22 gives rise itself to no callosal fibres. It would also be in keeping with our tentative summary of the experimental data which suggests that the major outflow from the primary auditory cortex is to a region anterior to itself. The correspondence between the two sets of data is at best rough but is close enough to suggest that further research may clarify this problem.

I will close this section with the consideration of a hypothetical problem. Could one develop pure word-deafness in one ear? The extent of duplication in the auditory pathways would almost ensure that the lesions necessary to produce this in a patient could hardly occur as the result of natural causes. With more detailed knowledge of the anatomy of the system one could probably specify what the requirements of such an unlikely lesion would be. Hartmann (1907) thought that one of his patients showed this phenomenon. There are, however, so many difficulties in the interpretation of other data pertaining to this particular case that I prefer to suspend judgment on the possibility of such a unilateral word-deafness.

4. *Lesions of Wernicke's Area*

Pure word-deafness as the preceding discussion suggests probably results

from the disconnexion of Wernicke's area from auditory stimulation. The normalcy of the patient's speech testifies to the intactness of Wernicke's area. With a lesion in Wernicke's area proper not merely is verbal comprehension impaired, but speech is also impaired. I will not present my conception of this type of aphasia extensively here but would only point out that the loss of Wernicke's area can be regarded as the destruction of a memory store – as it was in fact regarded classically. Presumably it functions importantly as the 'storehouse' of auditory associations. I have already suggested the importance of the angular gyrus in acting as a region involved in cross-modal associations, particularly in cross-associations between either vision or touch and hearing. If the angular gyrus is important in the process of associating a heard name to a seen or felt object, it is probably also important for associations in the reverse direction. A 'name' passes through Wernicke's area, then via the angular gyrus arouses associations in the other parts of the brain. It is probably thus that Wernicke's area attains its essential importance in 'comprehension', i.e. the arousal of associations.

I have presented this only cursorily since a more extensive discussion would lead us to a consideration of topics lying beyond the range of our interest at this point. I would like to stress that what is here regarded speculatively as the function of Wernicke's area implies the existence of extensive connexions to the angular gyrus region. Since this latter region is probably so poorly developed in subhuman forms, the fuller knowledge of this aspect of the connexions of Wernicke's area depends on careful study of those rare human cases with small lesions in the first temporal gyrus, particularly in its posterior portion. It is hoped that such studies will be made in the near future.[14]

5. *Tactile Aphasia*

This term describes a disturbance characterized by an inability to name objects tactilely with preservation of the ability to name on the basis of visual or auditory stimulation and in the presence of intact spontaneous speech. The existence of this condition has been disputed (*see* for example the discussion in Critchley, 1953). The case of Geschwind and Kaplan (1962b), however, established beyond doubt the existence of this entity and I will therefore present the relevant findings in this patient. I will confine myself to this aspect of the patient's problem and reserve dis-

cussion of the patient's 'apraxic' disturbances until a later section of the paper.

This patient had had an excision of a left frontal glioma. We examined him about six weeks later. This patient, when blindfolded, incorrectly named objects placed in the left hand. That this defect was one of *naming* was proved by several facts: (1) the patient would handle the objects correctly in the left hand while he was giving an incorrect name; (2) if the object was taken away and the patient was then instructed to select the object he had held from a group, he always selected the correct object either visually or tactually with his left hand; (3) similarly he could, after holding an object, concealed from vision, draw it correctly with his left hand although he had misnamed it. By contrast, after holding the object while blindfolded in his *left* hand, he could not afterwards select it from a group or draw it with the *right* hand. He correctly named objects held in the right hand and could draw such objects or select them from a group with the *right* hand but failed if he attempted to use the *left* hand for these tasks. That the disturbance was not one of transfer between limbs but rather between hemispheres was shown by the fact that he could draw with the left foot a pattern drawn on his left hand but not one drawn on his right hand.

Testing of elementary somesthetic sensation was difficult to carry out in the left hand if verbal responses were demanded but not if nonverbal responses were used. Thus, he demonstrated correct position sense on the left when he was made to respond by pointing up or down with the left hand; verbally his answers were random in this situation. Two-point discrimination on the left was normal when tested by having the patient indicate with one or two fingers the number of points touched. By contrast his verbal responses were random; not only were replies of 'one' and 'two' given incorrectly, but such totally inappropriate responses as 'four' or 'eight'. He could correctly point with his left hand to a place touched on the left side but gave incorrect verbal responses. Pain sensation similarly could be shown to be normal.

In brief this patient responded correctly to somesthetic stimulation if response was demanded from the same hemisphere as the stimulus but not if response was demanded from the opposite hemisphere. Thus, the patient responded correctly with his left hand to somesthetic stimulation of the left side of the body. By contrast his responses to such stimulation

with the right hand were incorrect. In addition his verbal responses, which of course would have had to come from his left hemisphere, were incorrect when he was given somesthetic stimulation to the left side of the body. By contrast he responded correctly with the right hand to somesthetic stimulation of the right side of the body and gave correct verbal responses to such stimulation; in this testing situation he gave incorrect responses with the left hand.

We interpreted these disturbances as reflecting a failure of somesthetic stimulation to cross to the opposite hemisphere and though that we would probably find a callosal lesion. The post-mortem confirmed the presence of a callosal infarction, probably secondary to ligation of the left anterior cerebral artery at the time of excision of the left frontal lobe. Tumour was entirely confined to the left hemisphere and did not involve either the callosum or the right hemisphere.

Had similar cases been observed before ours? Liepmann and others of the writers about the turn of the century had already commented on the inability of a patient to imitate with one hand the postures of the other as reflecting a callosal disconnexion. In addition. Liepmann (1900) called attention to the fact that the Regierungsrat who gave poor verbal responses on somesthetic stimulation must have had nearly intact sensation as evidenced by nonverbal manifestations. This disturbance was due to a disconnexion *within* the left hemisphere rather than to a callosal lesion. Goldstein (1908, 1927) on the basis of his own experience thought that a callosal lesion caused astereognosis on the left side of the body. Critchley (1953) mentions several other authors echoing the same opinion. Goldstein thought that this was the result of the fact that the left hemisphere was dominant for sensation. A more likely explanation is that Goldstein misinterpreted the incorrect verbal responses of his patient as representing sensory loss; he did not check whether sensation was intact when nonverbal criteria were used. The case of Trescher and Ford (1937) was regarded as having a 'tactile agnosia' on the left. Their patient showed only an inability to identify letters but not objects placed in the left hand. This more limited disturbance may well have a somewhat different interpretation from the more extensive disturbance in our patient.

The findings and interpretation of Geschwind and Kaplan have been more recently confirmed by Gazzaniga *et al.* (1962) who were able to

demonstrate similar disturbances in a patient with a surgical transection of the corpus callosum.

None of these cases permit a more precise delineation of the pathways involved; one can only conclude that they traverse the midcallosum, a result already likely on anatomical grounds and on the basis of experimental results (Myers, 1962b). My earlier discussion on the somesthetic system in animals makes it likely that Flechsig's principle is followed here and that there are no callosal fibres from the primary somesthetic cortex in primates; the same rule probably holds in man. Ettlinger's (1962) experiments involved so much of parietal lobe posterior to the postcentral gyrus that they do not help us in deciding whether a more or less circumscribed part of the parietal lobe comprises the association cortex from which the callosal fibres which transfer somesthetic stimulation to the opposite side originate. After synapse at the corresponding locus in the left hemisphere the 'message' presumably can be shunted to the speech area (i.e. the auditory association cortex of area 22) or to other parts of the hemisphere.

This simple model which is concordant with the known anatomical facts has certain interesting implications. A lesion of the right parietal lobe which involves the association cortex might produce the same effect as a callosal lesion, i.e. a defect in naming objects held in the left hand and a failure of the right hand to select or draw correctly objects held in the left hand. It is possible that this syndrome exists although the lesion producing it probably must be a large one. It is also likely that such cases have been incorrectly recognized as cases of astereognosis rather than cases of tactile aphasia because of failure of correct examination technique. The problem of the locus of the lesion producing astereognosis has long been a moot one and many authors have suggested a posterior parietal localization (*see* discussion in Critchley, 1953). Perhaps those with posterior parietal lesions were in fact cases of tactile naming defect based on the disconnexion of somesthetic regions from the speech area.

A lesion of the somesthetic association cortex on the left might have a more extensive effect. By destroying the connexion between left somesthetic cortex and speech area it should lead to a failure of tactile naming in the right hand. The lesion could also destroy the terminus of callosal fibres from the right hemispheric somesthetic association cortex and could therefore also produce tactile naming defect on the left. The net result

should be a bilateral tactile naming defect. There is some evidence for the existence of this condition. My colleague, Mrs. Edith Kaplan, has recently called my attention to a patient who showed a marked difficulty of tactile naming in both hands while naming *visually* was nearly normal. That the disturbance was one of naming was shown by the fact that the patient could handle the object correctly or could select it afterwards from a group without error. This patient showed a further additional feature; he could correctly select from a group with one hand an object held with the other hand. This suggests that the callosal connexions between the two somesthetic association cortices were intact and that the lesion must lie between left somesthetic association cortex and speech area.

This patient exhibited no aphasia in speaking and an occasional mild visual naming difficulty. His chief finding, other than the tactile naming disorder, was pure alexia with agraphia. The evidence appeared good that the lesion was in the left posterior parietal region but in the absence of confirmatory evidence, we must restrict ourselves to the fact that this case illustrates the possibility of a bilateral disturbance of tactile naming in the presence of a much milder visual naming difficulty.

Other cases described in the literature are almost certainly cases of the same disturbance although again comparison is made difficult by the failure of most authors to have tested for evidence of retained stereognostic function by nonverbal means. Cases such as those of Foix (1922) in which a unilateral lesion is said to have led to bilateral astereognosis might well have turned out to be cases of bilateral tactile aphasia had tests for nonverbal recognition been employed. Some others have preferred the term 'tactile agnosia' for such cases as those of Raymond and Egger (1906). I feel, however, unconvinced by Claparède's highly philosophical critique of the use by these authors of the term 'tactile aphasia'. The broader question of the position of the agnosias will be dealt with in the next section.

IV. THE AGNOSIAS

The agnosias have a.ways represented an unusually difficult group of disturbances. Classically they were defined as disturbances of 'recognition' without disturbances of elementary sensation. Some observers have simply denied the existence of disturbances of this type. There has been the attempt on one side to reduce them to perceptual defects of complex

nature, such as continuously shifting thresholds. On the other hand there has been the tendency to deal with such disturbances in terms of rather complex psychological malfunctions such as loss of ability to perceive Gestalts. A more classical approach was that of Lissauer (1889) who divided agnosias into apperceptive and associative, or disorders of primary and secondary recognition. The theory that I will present here will propose that the agnosias are indeed associative disorders; what will be added to the classical notion will be an analysis of just what type of associative disorder is involved and an explanation of certain clinical features that have in the past acted as stumbling blocks to analysis of these disturbances. I have already discussed in detail the agnosias of animals. The human cases are also based on disconnexion but I hope to show that the mechanisms are different in important ways from those operative in animals.

Workers in this field have tended to use a certain set of criteria for calling a disturbance an agnosia. First it is necessary to show either (1) that elementary sensation is intact or (2) that if an impairment of elementary sensation is present it is not sufficiently severe to explain the disturbance of recognition. Assuming this to have been demonstrated, how has it been possible to demonstrate that the failure of recognition was not simply a failure of naming? There have been several criteria generally employed. I will list them first and discuss their usefulness afterwards:

(1) *The 'agnosic' disturbance is a circumscribed one.* There is a common view that aphasic misnaming cannot be confined to a single modality; even more strongly it is assumed that it cannot be confined to a class within a modality. From this point of view an aphasic misnames everything; by contrast there can be a visual agnosia or even an agnosia for colours alone.

(2) *'Agnosic' errors differ in character from 'aphasic' errors.* – It is often assumed that aphasic errors are of one type, agnosic of another; the aphasic error is a 'linguistic' one, the agnosic a 'perceptual' one. According to this view, 'chair' for 'table' is an aphasic error but 'a small package' for 'a book' is an agnosic error; 'something to eat with' for 'spoon' is an aphasic error but 'pencil' for 'knife' is an agnosic error.

(3) *The 'agnosic' cannot choose the correct response.* – The aphasic who has failed to name is usually described as being readily able to accept the correct name when it is offered. The 'agnosic' by contrast is said to be incapable of selecting the correct name.

(4) *The 'agnosic' describes his perceptual difficulty.* – The aphasic is usually pictured as saying, "I know what it is but I can't find the name." When asked about the appearances of objects he asserts that they appear normal to him. The 'agnosic', by contrast, informs the examiner that things look different to him or that he has difficulty in seeing clearly. Alternatively he may offer dramatic descriptions of bizarre perceptions, thus apparently providing direct evidence that he is experiencing distortions of his perceptions.

(5) *The 'agnosic' cannot show the use of the object.* – This criterion is actually the most complex. The aphasic is said to be able to show the use of the object. The 'visual agnosic' cannot show the use of the object but can manipulate it correctly if it is actually placed into his hand. The 'apraxic' according to this classical analysis cannot handle the object correctly even when it is placed in his hand.

As I hope to demonstrate, all of these criteria are actually inadequate for the separation of patients into two classes of distinct physiological disturbance. The fundamental difficulty has been in the acceptance of a special class of defects of 'recognition', lying somewhere between defects of 'perception' and of 'naming'. What indeed are the criteria for 'recognition' and is it a single function? I believe in fact that there is no single faculty of 'recognition' but that the term covers the totality of all the associations aroused by any object. Phrased in another way, we 'manifest recognition' by responding appropriately; to the extent that any appropriate response occurs, we have shown 'recognition'. But this view abolishes the notion of a unitary step of 'recognition'; instead, there are multiple parallel processes of appropriate response to a stimulus. To describe the behaviour correctly we must describe the pattern of loss and preservation of responses to each particular type of stimulus.

Let us make this more concrete by referring to a particular patient's behaviour. I will return for my example to the case of Geschwind and Kaplan (1962) to which I have several times made reference. Let us consider his difficulty in identifying objects placed in the left hand. He incorrectly named objects placed in the left hand. He could, however, draw the object afterwards with his left hand or select it from a group tactilely or visually. It is obviously correct to describe this patient as showing correct identification by nonverbal means and incorrect identification verbally of objects held in the left hand. It is difficult to see how this could

in any way be described as a 'perceptual' disorder since perception must have been intact to insure correct selection from a group or the drawing of the object.

We have interpreted this disturbance as a result of disconnexion from the speech area. In addition the patient showed other disturbances of identification not due to disconnexion from the speech area. Thus, he could not select or draw with the right hand an object held with the left, concealed from vision, or select or draw with the left hand an object held with the right. But again 'perceptual' disturbance in either hand is excluded by the patient's ability to select or draw with a given hand an object that had been held in the same hand. We can only regard the defect as associative – what was perceived by the right hemisphere could not be relayed to the left and vice versa.

Let us return for a moment to the failure to identify verbally with the left hand objects held in that hand which we have attributed to disconnexion from the speech area. It may be argued that we have evaded the fundamental issue, that although we have shown that the patient could not name the object placed in his left hand but yet could draw it or select it afterwards from a group, we have not really shown whether the patient had recognized the object. This question is based on two assumptions which I believe are incorrect. The first assumption is that 'the patient' is an entity. But in someone whose two hemispheres operate as independently at this patient's the word loses its ordinary meaning. There is no answer to 'Did the patient recognize'? There are, however, clear answers to 'Under what conditions did the right or the left hemisphere recognize'?

The second assumption is that recognition is more than the sum of the individual acts of naming, drawing, handling, selecting, etc. If it is more than these, how would one test for this faculty and how would one show that this test established some superordinate function of recognition? I do not know of an answer to this question.

I have argued that the patient's failure to identify verbally objects held in the left hand must be regarded as a result of separation of the right somesthetic region from the speech area and can in no sense be regarded as a perceptual defect. In the light of this analysis, let us now consider the patient's responses from the point of view of the classical criteria listed above which were used to distinguish 'agnosic' from 'aphasic' disturbances. This analysis will demonstrate the inapplicability of these criteria.

(1) *Our patient's disturbance was highly circumscribed* to one modality and indeed one-half of the body. Classically such circumscription was often regarded as being incompatible with an aphasia. Yet as soon as one admits the substantial restriction of speech to one hemisphere, the possibility of cutting off one-half of the brain from speech must be considered. There is no basis therefore for this classical distinction.

(2) *The errors made by our patient were not 'errors of naming'* in the sense of the commonly used criteria. He could call a 'screw-driver' a 'rubber band', a 'coin' a 'cigarette lighter'. These are not (without meaningless extension of the term) errors in sphere nor are they errors based on similarity of sound. They are, therefore, not 'aphasic' errors according to the usual criterion. But it is easy to see that if a particular part of the brain is fully disconnected from the speech area, there is no reason that an erroneous name should be related in any way to the correct term. The more complete the disconnexion, the more random the errors must be. The less complete the disconnexion, i.e. if some information gets through to the speech area, the more the errors may be related to the correct response. It should be added that in fact the classically anomic patient often makes naming errors not related by sphere or sound.

(3) *Our patient showed inability to choose the correct verbal response when it was offered to him.* – When he misnamed an object we would offer him several choices. The patient would generally not select the correct answer from the group offered. If, however, the speech area is fully disconnected from the right hemisphere, there is no reason why the speech area should select the correct term. It is obvious that the ability to select the correct word depends on there being some connexion between the site of perception and the speech area. The ability to select the correct word from a group when the patient cannot find it spontaneously indicates a lesser degree of disconnexion. Phrased in another, diagrammatic way, one could conceive that when one offers a word to a patient he in some way compares the images or memories aroused by this word with the sensations he is receiving from the object. If there is no site for such comparison, the patient will not recognize the correct word when it is offered. I would like to point out that in fact the obvious nominal aphasic often fails to accept the correct word when it is offered. Furthermore, even when the patient insists that 'I know what it is but I can't tell you the name' he may fail to accept the correct name from a group.

(4) *The patient gave descriptions of his 'perceptions'*. – Thus, when holding a half-dollar, the patient said that it was a cigarette lighter. When asked if it had corners he said, "Oh, yes, there's one, there's another." He would describe incorrectly the object held in his hand in most instances. The same objection applies here as to the previous categories. If the patient's speech area is disconnected from a site of primary perception why should his speech area be able to describe what is going on at the site of the primary perception? Clearly, it should not. The fact that 'the patient' (i.e. the speech area) gives a description does not mean that we are getting an actual description of the perceptions going on in another disconnected part of the brain. We must again remember that we are dealing with more than one 'patient' here. The 'patient' who speaks to you is not the 'patient' who is perceiving – they are, in fact, separate.

(5) Our patient lacked only one of the classical criteria of 'agnosia'. According to these classical criteria, a patient with 'tactile agnosia' should mishandle objects which he has not seen but should respond correctly to the sight of the object (i.e. his behaviour should be opposite to that of the visual agnosic). Our patient in fact handled objects perfectly correctly when blindfolded. It will return in a later part of my discussion of the agnosias to this question of the use of objects.

1. *The Problem of Confabulatory Response*

I have discussed in some detail a disturbance which might have been called 'agnosic' and tried to show that this disturbance is much more meaningfully described as a naming defect resulting from disconnexion from the speech area. There are certain further implications of this interpretation which I would like to present now. One most important implication is that the 'introspections' of the patient as to his disability may be of little or no use to the examiner. The patient cannot 'introspect' about the activities of a piece of brain which has no connexion to the speech area. What he tells you is of little value in elucidating the mechanism and may indeed be actively misleading. Indeed, it becomes clear that many of the patient's responses can only be described as confabulatory, i.e. they are attempts to fill gaps in the information available to his speech area; phrased in more conventional terms they are attempts to explain what the patient cannot understand.[15]

These confabulatory responses have been a major source of confusion

for many years, particularly in the assessment of difficulties of sensory identification. It is curious that sophistication concerning confabulatory behaviour has been much more widely developed in certain areas than others. When the patient with a severe recent memory defect asserts that the examiner spent the previous evening with him in a bar, we neither ask ourselves why the patient had hallucinations of seeing the examiner the night before nor why he is now having false memories. We see this instead as the verbal filling in of a gap. We discount the attribution by the same patient of his lack of knowledge of current events to the fact that he has never been interested in politics. Similarly, if an aphasic patient tells us that his trouble in speaking is the result of his ill-fitting dentures or a sore throat, we do not take the attitude that he is giving us any very useful insight into his illness but rather that he too is attempting to explain a gap in his performance. An even more extreme example is that of patients with denial of blindness who may be prepared to describe in detail many of the objects in the room; once we know that the patient is blind, we do not attempt to make use of his 'introspections' about his visual perceptions. We do not take seriously this patient's protestations that he has failed in some task because the light is poor or he is not wearing his glasses. Furthermore, we do not assert as an alternative that the patient is really hallucinating.

The 'higher' the function impaired, the more readily the patient may succeed in fooling others with confabulatory responses. I recall one patient with pure alexia without agraphia who asserted that his trouble in reading was due to his 'blindness in his right eye'. The resident staff correctly realized that he meant a right visual field defect when he spoke of a blind right eye. However, they incorrectly accepted the patient's attribution of his reading disturbance to this visual impairment. Only when the discrepancy between reading and object-naming was pointed out to them did they realize that they had too readily accepted the patient's 'explanation' of his disability.

There are many occasions in which patients with aphasia, dementia, blindness, or recent memory defects produce confabulatory responses which are recognized. There are probably equally numerous situations in which such confabulatory responses are not recognized as such by the examiner. Most of the conditions called 'agnosias' fall into this category. Much time has been spent in the vain attempt to analyse the supposed

disorder of perception which has been regarded as underlying the patient's responses.

In summary, the theory here proposed is that most of the classical agnosias are highly isolated disturbances of naming which are the result of disconnexions from the speech area. The prominence of confabulatory responses may incorrectly lead the examiner to believe that he is dealing with a perceptual disorder.

2. *Inability to Identify Colours*

The patient of Geschwind and Fusillo (1964) (discussed in an earlier section of the paper) showed pure alexia without agraphia and a failure to identify colours. This disturbance could readily have been called an 'agnosia for colours'. Yet, analysis of his responses makes it clear that his difficulty could be correctly described as a disturbance in the naming of colours. By all nonverbal criteria he identified colours correctly – he matched and sorted correctly, did the Ishihara test perfectly and even properly matched colours to pictures of objects, yet he failed consistently in verbal naming. It is obviously appropriate to describe his disturbance as a colour-naming defect since by no criterion could he be shown to have a perceptual defect. His pattern of responses also illustrates the lack of usefulness of the classical criteria. Thus he exhibited a very circumscribed naming defect. Only once in many testing sessions did he misname an object, yet his colour-naming remained consistently extremely poor. He generally failed to accept the correct name of the colour when it was offered. Indeed, when we told him, for example, that something was not grey but red, he said, "Well, it looks grey to me – maybe it's a reddish-grey." On another occasion he said, "You say it's white but it looks tan to me." Again the most parsimonious assumption is that he had a colour-naming defect with confabulatory response. Whenever we asked the patient to give a *verbal* account of the colour experiences of his right hemisphere, he produced a confabulatory response; if we tested the right hemisphere *nonverbally*, we got evidence of perfectly normal colour perception.

I would like to discuss the one instance of poor matching by this patient since the careful analysis of this failure was most illuminating. The patient was given a pile of colour chips which contained chips of two different shades of each of the primary colours. He was shown a chip of

one of the two shades of red and told, "This is red. Now pick up all the red chips." He picked each chip up slowly and examined it. He finally separated out all of the chips of the selected colour as well as all the chips of one of the two shades of green. When asked what colour the chips were which he had selected he said, "These are red and these others (indicating the green ones) are a different shade of red." He was then able to separate these chips into two piles. When shown a chip of the *other* shade of green and asked which of the two piles it was most like, he unhesitatingly classified it with the pile of green chips.

There was clearly a sorting error here but full consideration of the patient's behaviour seems to exclude the possibility that we are dealing with a perceptual error. I believe the sequence of events to have been as follows. We had asked the patient to "pick up all the red chips," rather than to "pick up all the chips like this one." He had done the task slowly probably because he did it by picking up each chip and naming it. Having misnamed a chip of one of the shades of green as red, he had then selected the remaining chips of this shade. We may consider this as a sorting error resulting not from failure of discrimination or perception (which his subsequent behaviour in this task excluded, i.e. the rapid separation of the two colours) but as a 'secondary sorting error' which resulted from doing the task verbally. Thus, even failures in sorting should be analysed carefully to determine whether they are secondary to misnaming or are truly failures of discrimination.[16]

3. *Classical Visual Agnosia*

There are many remarkable accounts of this disturbance in the literature (e.g. Lissauer, 1889; Lange, 1936; Brain, 1941; Macrae and Trolle, 1956; Ettlinger and Wyke, 1961; Hécaen and Angelergues, 1963). In going through these accounts one is immediately struck by the frequent evidence of preservation of nonverbal visual identification while verbal identification is impaired. One striking feature of many of these cases is the fact that the patient's ordinary behaviour is in marked contrast to the supposed perceptual disturbance. Thus, one may read an account of a patient who cannot 'identify' a glass of water and yet a few minutes later picks it up and drinks from it. Much stress has been laid on the fact that a patient may show normal behaviour in a 'natural' situation but not in an 'artificial' one. Once one abandons the notion of a unitary process of 'recognition',

it is easy to see that the patient may fail to 'identify' an object, i.e. fail to give a verbal account of what is going on in his visual receptive regions and yet be able to respond nonverbally to a nonverbal stimulus. Most of the pathways involved in the nonverbal response may be quite different from those involved in naming. For a thirsty man to respond appropriately to the sight of a glass of water by grasping it and drinking from it is *anatomically* a different task from that of responding to a verbal request for identification. It is certainly reasonable that a naming defect should not produce gross functional impairment; by contrast it would be very difficult to understand how a 'higher-order perceptual defect' could leave ordinary perceptual function unimpaired. A similar preservation of function appears in those cases who can draw the object which they cannot recognize (Lange, 1936) a behaviour obviously compatible with a naming difficulty.

The patient of Ettlinger and Wyke (1961) made many errors in visual naming (e.g. 'a box' for 'a glove', 'for cooking' for 'a spoon') and many fewer in tactile naming. This case can be used as further evidence against the idea that an aphasic naming disorder must cut across modalities. The data of these authors show that while 'agnosic' errors (e.g. 'box' for 'glove') were more common visually than tactilely, the same was true for 'aphasic' errors (e.g. 'for cooking' for 'a spoon'). A striking feature of this case was the matching of outline shapes without error even though the patient made nine errors in naming outline pictures of ten objects. Certainly a naming defect is the simplest explanation of this disorder. This case was similar in this respect to the earlier case of Brain (1941).

The remarkable case of Macrae and Trolle (1956) presents another example of one of these striking discrepancies. The authors point out that when the patient was pressed with regard to his method of finding his way to and from work, "he could not visualize the street plan or the route." Despite this he did in fact drive two miles a day to work without difficulty. I would suggest that 'visualization' here means either developing or describing an internal picture in response to a verbal stimulus – but this will not occur if the visual system is separated from the speech area. Yet, the subject may respond nonverbally to the actual route.

Patients with classical visual agnosias have often been presumed to be suffering from 'perceptual' disorder on the basis of their 'introspections'

concerning their illness. The case of Lissauer (1889) illustrates how little such 'introspections' are to be trusted. Thus, Lissauer comments that when the patient misidentified an object, he would, after learning its true nature, excuse himself by saying that "his eyes had deceived him at first." As Lissauer comments, this statement, coupled with the fact that some of the misidentifications were so curious, might have led the examiner to believe that the patient had been having sensory illusions of some type. But if the examiner then asked the patient whether the object, now that he knew what it was, looked any different from the way it had before, he received a vigorous denial. The patient in fact now asserted that the object had looked the same before but that "his poor memory was responsible for the fact that it had not occurred to him what the thing was called and what it was." Had Lissauer pushed his analysis somewhat further here he might well have been able to see that the 'misidentification' was actually only misnaming. Lissauer himself comments elsewhere in the paper, "The patient succeeded ... in drawing simple objects which he didn't recognize, a striking proof that he was well able to perceive their forms." Similarly, although the patient failed to name colours, he succeeded without any difficulty in matching tasks.

4. *The Lesions of Classical Visual Agnosia*

At first it would be tempting to ascribe classical visual agnosia simply to disconnexion of the visual region from the speech area. This interpretation, however, runs into obvious difficulties. Thus, we have already explained in an earlier section that pure alexia without agraphia (with its frequent accompaniment of colour-naming difficulty) is the result of disconnexion of the visual cortex from the speech area. In these cases there is usually destruction of the left calcarine cortex. In addition there is a lesion of the splenium of the corpus callosum which isolates the right visual region from the speech area. It was particularly noted that these patients did not show difficulties in object-naming and often showed retained ability to read numbers. I said in the earlier discussion that it seemed likely that the explanation for the preservation of object naming was that objects could arouse somesthetic and other associations more anteriorly in the right hemisphere; these would then traverse the callosum in the preserved portions anterior to the splenium and thus reach the speech area. For the development of a naming defect for objects, failure to arouse these tactile

associations (a visual-tactile disconnexion) is a necessary addition to the disconnexion of visual regions from the speech area.

The lesions found in the majority of cases of visual agnosia support this notion. Most commonly observed are very large bilateral parieto-occipital lesions. There are probably no direct connexions between the visual and the somesthetic cortex so that visual-somesthetic connexions must be mediated via the intervening association cortex. A large parieto-occipital lesion would effectively cut off such connexions. One can also see the reason for the rarity of classical visual 'agnosia'. If the lesion is too small, a large enough extent of association cortex is left to make the defect a minor one. If the lesions are too large, the patient may show gross aphasic impairment as well as marked disturbances of visual-motor connexions. In this case his impairment of speech function may make analysis exceedingly difficult. The cases with very extensive lesions might with some justification be regarded as manifesting disturbances of the general ability to manifest visual 'recognition' since all outflow channels may be cut off except possibly some primitive and not very extensive pathways descending subcortically.

There is another but much smaller group of visual 'agnosias' who have unilateral lesions. This group shows destruction of the left visual cortex. In addition there are always lesions of other structures. One group of these patients shows involvement of the splenium. Others have such extensive involvement of the white matter or of the association cortex in the left occipital and parietal lobes as to have destroyed either the fibres coming from the splenium or their terminations in the left hemisphere (Lange, 1936; Nielsen, 1937).[17] The beautifully described case of Lissauer (1889) turned out to have such a lesion (Hahn, cited by Meyer, 1895–1896) although he himself had expected bilateral lesions.

Why do these cases with unilateral lesions manifest more than pure alexia? It is difficult to be certain and I would hope that more careful study of cases in the future may provide the answer. One possibility is that in some people visual stimuli in the right hemisphere fail to arouse tactile associations or do so weakly. These patients should therefore develop a more extensive naming defect when the right visual regions are cut off from the speech area. Another possibility is that in these cases the lesion in the splenium extends further forward than it does in the cases of pure alexia without agraphia. The lesion would thus cut off not only fibres from the

visual association cortex but also fibres from the somesthetic association cortex. In the case of Trescher and Ford (1937) the surgeon is said to have divided the posterior half of the corpus callosum; it seems likely that the lesion must have involved more than the splenium. The description of the case would suggest that the patient in addition to being alexic in the left half-field, also could not name objects in that field. This case therefore provides suggestive evidence for the hypothesis that lesions extending somewhat anterior to the splenium may cut off impulses from the somesthetic association cortex and thus prevent visual stimuli from reaching the speech area via this detour.

5. *The Handling of Objects*

It was pointed out earlier that one of the classical criteria for a visual agnosia was failure of the patient to demonstrate the use of objects shown to him. It was pointed out that according to this scheme the 'aphasic' could show the use of an object shown to him; the 'visual agnosic' could not do this but could manipulate the object correctly if it was placed in his hand; the 'apraxic' could not perform even with the object in his hand. I have reserved this criterion for special discussion since it appears at first glance to provide a very reasonable basis for separating disturbances of recognition.

This criterion, however, also fails as the mark of a separate category of 'recognition' disturbances. In the first place, as I have already commented, some patients who are severely 'agnosic' by the other classical criteria listed earlier can in fact respond correctly to the sight of objects. If one simply interpreted agnosias as deficits of 'recognition' there should be no reason why correct demonstration of the use of a seen object should be preserved in the presence of a severe disturbance of this type. Furthermore, as I will show in the section on motor disturbances, there are severely apraxic patients who fail to respond to the sight of an object yet handle the actual object correctly when it is placed in the hand. By the classical criteria these patients should be called agnosic. Yet these same patients may be able to name the object and describe its use, behaviour certainly incompatible with a loss of 'recognition'.

As I will point out in the section on the apraxias, they are also the result of disconnexions. The lesions producing these disturbances separate sensory from motor regions. It is obvious that the kind of lesion which

causes classical visual agnosia will frequently although not invariably result in the disconnexion of the visual regions not only from the speech area and from the somesthetic areas but also from the motor region.

This discussion illustrates again the principle that division of phenomena into aphasic, agnosic or apraxic often obscures underlying mechanisms. It is far better in general to specify the classes of response which are lost or are preserved in response to each class of stimuli.

6. *The Conditions for Confabulatory Response*

I have advanced a theory of classical 'agnosic' disturbances which views them as naming disturbances which are frequently overlaid with confabulatory responses. Why do these confabulatory responses occur? Weinstein, Cole, and Mitchell (1963) have stressed certain features of this type of response. Let me paraphrase some of their major conclusions: (1) Confabulation does not occur in the absence of a defect; it is, indeed, clearly an attempt to fill in a gap of performance of some type. (2) Confabulation is less marked in the presence of aphasia; this seems reasonable since it clearly is a response of the speech areas. (3) Confabulation is more likely in the presence of some over-all impairment of awareness, such as generalized dementia or clouding of awareness.[18] To these three conditions I would add a fourth (Geschwind, 1963b): Confabulation is much more likely in the presence of disease of association cortex or association fibres (either commisural or intrahemispheric) than it is in cases of damage of the primary sensory pathways up to and including the primary sensory cortex. We can combine this with the third condition above to make the following statement: The more demented or clouded the patient, the more likely hes is to show confabulatory response in the presence of any defect of whatever origin; the less dementia and clouding are prominent, the more confabulation depends on lesions of association cortex or fibres; the two mechanisms interact frequently. These, of course, are not the only conditions; the influence of personality prior to illness may also be highly important.

Many examples come to mind to support this contention. My personal experience of several cases of denial of blindness illustrates this rule. The first case was that of a man suffering from a septicæmia, markedly febrile, drowsy and confused, who was blind as the result of infection of the anterior chambers of both eyes. Another was a man with a large sub-

frontal meningioma with advanced optic atrophy and a classical severe frontal syndrome with dementia. In these cases denial of blindness was related to marked general impairment of function of the nervous system. I have seen a confused patient who gave bizarre confabulatory responses when asked to name objects held in the hand. He could not, however, select the objects correctly from a group afterwards. This therefore must have represented the confabulatory response to an actual sensory loss. The same patient gave confabulatory responses to visual field testing when there was no stimulus in the field but gave correct answers to stimuli within the bounds of a normal visual field.

In contrast with these cases, patients with bilateral occipital infarcts (which probably usually involve much visual association cortex) are likely to manifest denial of blindness without marked dementia. Blindness from peripheral lesions is far less likely to produce such confabulatory denial in the absence of dementia or clouding. Confabulatory denial of a lost limb is uncommon; although the patient frequently has a phantom limb, he nearly always treats it as a phantom. But denial of hemiplegia is common and probably more common in the presence of accompanying lesions of association areas; even if it were insisted that all of these patients show some dementia or alteration of awareness, it is obvious that it is far less than the degree of dementia needed for the denial of a peripheral defect. I have, of course, already cited many examples of confabulatory response in the earlier sections of this part of the paper.

There is some suggestive but not definitive experimental evidence for this hypothesis. Warrington (1962) carried out experiments on visual completion in patients with hemianopias. She found that those patients who were unaware of a hemianopia were likely to 'complete' forms across a field defect while those who were aware of their hemianopias did not. Thus, the first group would report a segment of a circle in the good field as 'a circle' while the second group would report it as an incomplete circle. She found that this tendency to complete forms across field defects was strongly associated with the presence of a parietal lobe lesion, i.e. in our sense a lesion of association cortex. The anatomical and other data are however not fully convincing. Thus, of the 11 patients who showed completion, 6 had tumours and 3 of the remaining 5 showed mental deterioration. Thus, 9 of 11 had evidence either of widespread deterioration or of a lesion likely to cause more than focal disturbance. (It must be admitted,

however, that the six tumours were all parietal in location.) In the group of 9 patients with little completion only 2 had tumours (neither of them parietal) and only one other case of this group was said to have shown deterioration. Thus, only 3 of 9 patients in this group had evidence of dementia or of a lesion with possible widespread effects. It is therefore possible to use Warrington's data in support of a different thesis from the one I have advanced – that patients with dementia or tumours are more likely to develop unawareness of a hemianopia and to complete forms across a field defect. Further studies of this type with greater description of the degree of dementia should help to resolve the issue in favour of one or the other interpretation.

Another paper by Kinsbourne and Warrington (1962) is also suggestive evidence for the thesis I have presented. They found patients with right hemisphere lesions who tended to misread the left halves but not the right halves of words ('novel' for 'level', 'cucumber' for 'encumber'); 4 of these cases showed complete left hemianopias but 2 did not. The authors thought that all of these patients had right parietal disease. It would be most interesting if it could be shown that this type of confabulatory reading error resulted from right parietal association area lesions, but this would require a control group of patients matched for dementia and with proven nonparietal right-sided lesions who would not show the defect.

Why should such confabulatory responses be less common in lesions of primary sensory cortex and more common in lesions of association cortex? I cannot answer with assurance, but I can propose a highly hypothetical mechanism which is at least suggestive of how such a situation could arise. Since the speech area has in the normal no direct contact with the visual cortex proper, destruction of the calcarine cortex leaves the speech area still innervated by visual association areas. The association areas in this case receive no stimulus from the visual cortex and send the 'message' to the speech area that there is no visual 'message', i.e. that all is black. This presupposes that the association areas never fail to send a 'message' to the speech area and that they always send *positive* messages of light or darkness. The destruction of association cortex or fibres corresponds to a totally unphysiological state – one in which *no* message is received by the speech area. Since this is not interpreted (not being one of the normal states) the speech area begins to react to random messages received from subcortical pathways or indeed responds to its own spontaneous firing. It

may respond to *incomplete* information coming over subcortical pathways which are inadequately extensive to carry all the information about the seen object; this may lead to errors which are less bizarre than those in which no information at all gets to the speech cortex. As I have stressed this is a highly speculative mechanism, but certainly not an impossible one.

7. *The Problem of Right Parietal Dominance*

It may be useful to speculate briefly on another problem of major interest in the current neurology of the higher functions, the question of the dominance of the right parietal lobe in spatial functions. This problem has been a perplexing one, perhaps because there is said to be a range of disturbances from right parietal lesions unlike those seen with left-sided lesions. On the one hand there is the fairly clear-cut syndrome of inattention to the left side of space, manifested by a tendency to bisect lines to the right, a tendency in copying to omit features in the left half of the picture, neglect of the left side of the body as shown by failure to dress it or even denial of its existence. The corresponding syndrome of neglect of the right half of space seems to be less common. At the other extreme one sees patients with right parietal lesions with the grossest impairments of function apparently not confined to the left half of space. These patients may show gross disturbances in drawing figures or copying involving much more than neglect of the left half of the object and difficulties in dressing not confined to the left half of the body. A common argument has been that since such gross impairments are more common with right than with left-sided lesions, the right parietal region is the dominant one for spatial functions. However, there is a difficulty with this argument. In the first place the sheer loss of the left half of space in some right parietal lesions suggests that a dominant left hemisphere is being deprived of information from the right hemisphere. By contrast, if the right side were usually dominant then one should frequently see left parietal lesions with neglect of the right half of space; this picture is, in fact, less common than cases with neglect of the left half of space. Thus, in particular, drawings with the right side omitted are less common than those with the left half omitted.

I would like to offer a tentative resolution of this paradox. In the first place, let us consider the possibility that the main effect of a right posterior parietal lesion is to disconnect the right visual and somesthetic cortex from

the dominant left hemisphere. (Whether the effects are due entirely to disconnexion from the speech area, I would hesitate to say.) The left side of the body and of space is then 'lost'. The patient will then respond in many instances by using the technique of confabulatory completion that I have discussed above and for which I have already cited experimental evidence.

The confabulatory response may perhaps be responsible for some of the more severe syndromes with right parietal lesions. If the confabulatory response is very bizarre, then the patient must make a major readjustment of all of space in order to deal with his 'experiences'. For example, an occasional patient will express the feeling that he has two sets of left arms, one which he sees and another which he feels. If he tries to order this bizarre sensation according to his previous experience he must grossly distort his entire body image. It will be no surprise if he now has trouble dressing even the right half of his body. Similarly, if in copying a picture he has a bizarre confabulatory structure in the left half of the field, he may find it necessary to readjust the right half – albeit unsuccessfully, in order to make his copy satisfactory. As I have already noted, this is a very tentative suggestion which probably needs recasting in a clearer form but I believe it may have the kernel of a new approach to the problems of right parietal syndromes.[19]

Let us consider briefly the vexing problem of why a left parietal lesion less often produces neglect of half of space than does a right parietal lesion. In the first place the right hemisphere is probably always at a disadvantage in relation to the left since the pathway from any part of it to the speech area is probably at least one neuron longer than the pathway from the corresponding part of the left hemisphere (there must be at least one extra neuron to take the impulse across the corpus callosum). One extra neuron may be of little import when added to a short path but probably the extra degradation of the stimulus becomes more important the longer the route. Any 'detour' around a lesion used by the right hemisphere always suffers this extra disadvantage and therefore makes it likely that the right hemisphere will be more completely disconnected. If in addition the right hemisphere responds less well than the left to stimuli (see the earlier discussion on dominance), it is subject to a further disadvantage. Thus disease may simply aggravate the normal disadvantage of the right hemisphere in being further away and responding less well to stimuli.

There is another mechanism which may contribute to the special disadvantage of the right hemisphere. The speech area and other dominant areas of the left hemisphere conceivably might receive *some* sensory information directly from the left thalamus in the situation in which the parietal association cortex was destroyed. This short cut is however not available on the right; the thalami have no significant commissural connexions (the massa intermedia is of minor significance and in any case is very frequently absent) and therefore impulses from the right thalamus must still reach the cortex of the right side before crossing to the other side.[20]

According to this view of right-sided association area lesions, they are more likely to lead to disability and to confabulatory response than those of the left side because the normally poorer linkage of the right side to the speech area, and possibly to other 'dominant' areas of the left hemisphere, is further weakened by the lesions. This approach may contribute to further understanding of some other phenomena which occur more frequently in right hemisphere lesions. Thus, Hughlings Jackson (1880) thought that *déjà vu* was more likely to result from right than from left cerebral lesions. Bingley (1958) confirmed this result. Mullan and Penfield (1959) found that visual illusions and *déjà vu* phenomena arose predominantly from the hemisphere nondominant for speech. Cole and Zangwill (1963) also found a predominance of *déjà vu* from the minor hemisphere although the predominance was not quite as great as in Mullan and Penfield's series. Teuber *et al.* (1960) found seizures involving visual experiences in 15 patients in all; there was a definite preponderance of right hemisphere lesions. These authors cite the study of Hécaen and Badaraco who found that 14 of 16 cases with visual experiences had right hemisphere lesions.

I would suggest that perhaps this tendency of the minor hemisphere to give rise to such illusions is not the result of localization of emotion or memory in the minor hemisphere but another example of the increased tendency to confabulatory response in cases of lesions of the association cortex on the right side. The reasons for the right-sided predominance would be the same as those given above.

It is interesting to note what Hughlings Jackson (1880) said about this predominance of lesions in the right hemisphere in producing what he called intellectual aurae: "These 'dreamy states'... cannot be owing to an epileptic discharge. It would be a remarkably well-directed and distributed

epileptic discharge which would give rise to the exceedingly compound mental state of being somewhere else.... I submit that (such an elaborate physical state) is owing to but slightly raised activity of healthy nervous arrangements consequent on 'loss of control'... possibly of some in the *cerebral hemisphere opposite the one*,[21] which I believe to be nearly always the right, in which the discharge begins...."

I would like to cite one further recent piece of work to illustrate that it may be possible to reinterpret certain studies which apparently support the idea of right hemisphere dominance for certain functions. I have chosen only one result in one of the many important papers principally by Milner and Kimura published from the psychological laboratory in Montreal. Kimura (1963) used a test in which a subject was shown a preliminary series of picture cards. After this he was presented with a test series of 140 cards; he was to reply, 'Yes' if he thought he had seen the picture in the preliminary series, 'No' if he thought that he had not. The total score was the number of correct recognitions minus the number of false positive responses. The group with right temporal lobe lesions scored significantly worse on this task than the group with left temporal lesions. Further analysis of these data, however, showed the interesting fact that the number of correct 'yes' responses was almost the same for both groups but that nearly all the difference in scores was due to the large number of false 'yes' responses by the right temporal group.

Could this not be interpreted as another example of confabulatory response resulting from a lesion of right hemisphere association cortex rather than as a special deficit of the right hemisphere in the retention of unfamiliar material? Kimura gives two reasons why the patients with right hemisphere lesions cannot be interpreted as having "a general tendency to make false recognitions." The first argument is that in an auditory memory task the right-temporal subjects showed no more tendency than the left temporal to give false positives. But since I have already given several examples of confabulatory response confined to a *single* modality, this argument would not rule out that the major effect of the lesions was to produce confabulatory response only to unrecognized visual stimuli. The other argument is a somewhat stronger one. Kimura points out that in another task, the overlapping Nonsense Figures test (where immediately after being shown a nonsense figure tachistoscopically the subject is required to select it from a group of figures on a card) the right temporal

group performed more poorly than the left, despite the lack of any penalty for false positives. Inspection of the data shows, however, that the significance level on this test for differences between the post-operative right and left temporal groups is between 0.01 and 0.02 while on the recurring figures test the significance level for the difference between the groups was less than 0.001. Furthermore, the difference between the post-operative groups on the nonsense figures test was less than 2 items. By any standard this difference appears far less impressive than the tendency for the right temporal group to produce false positive responses on the recurring figures test. Even if one were to accept the result on the nonsense figures test it would argue for only a slight, if significant, superiority of the right as against the left hemisphere on this task; it would not support the idea that the right dominance for this activity was anywhere near as marked as left dominance for speech. In any case it is obvious that many further careful studies of the type carried on by the Montreal workers will be needed to settle this issue as to whether the right hemisphere manifests dominance in any sphere.

8. *Visual Imagery*

Related to the problem of the agnosias is the problem of loss of visual imagery. Wilbrand's (1887) monograph on psychic blindness actually concerned itself primarily with patients in whom visual imagery was lost. I have already commented briefly in discussing Macrae and Trolle's interesting case on the patient who could not 'visualize' a route but could travel it correctly. The word 'visualize' has two connotations. We may, as it were, see a picture in 'our mind's eye', and can then say that we are visualizing. At a simpler behavioural level we may describe an absent scene. A classical notion was that visualization was an activity of the visual association areas. The evidence for this is suggestive but by no means complete. The peripherally blind can indeed describe absent scenes and say that they have internal visual imagery. Would a patient with a pure destruction of the visual cortex proper bilaterally still retain imagery? The answer to this question is not known.

Loss of the ability to describe an absent scene might reasonably result from a disconnexion of the visual regions from the speech area, as probably was the case in MacRae and Trolle's patient. That this failure to give a verbal account need not be associated with a loss of visual memories is also

illustrated by the same patient who could in fact correctly drive to work although he could not verbally describe the route. Was this patient capable of developing inner pictures? There would be no way of finding this out by interrogation since if the region in which such pictures were taking place were disconnected from the speech area, we would be unable to get a verbal account from the patient. The question is not a purely academic one since the eventual development of more advanced physiological techniques may settle some of these questions without recourse to a verbal account from the patient. This would be analogous to recent work on dreams for which objective criteria are now being developed.

The question of dreams and their locus is related to that of waking visual imagery. Are dreams functions of the visual cortex or of the visual association areas? These problems of imagery and dreams deserve further study using both standard clinical testing procedures as well as some of the new techniques employed so effectively in the study of dreaming.

V. THE MECHANISMS OF THE APRAXIAS

1. *Disconnexion from the Speech Area*

The term 'apraxia' in its modern usage goes back to Liepmann's (1900, 1906) employment of the term in describing the classic case of the Regierungsrat. Despite the great complexity of the case, Liepmann devised such remarkably appropriate methods of testing and analysed his findings so adroitly that he was able to predict the patient's lesions. At the patient's death two years after the publication of the paper containing Liepmann's clinical description, the post-mortem confirmed his predictions; subsequent study of the material in whole-brain sections established in detail the correctness of his ideas. The case has often been criticized as having too many lesions to be useful. It is difficult to understand this point of view when it is appreciated that the prediction of the loci of all the lesions was made in advance despite the fact that no similar patient had previously been described. These papers therefore differ importantly from those in which the correlation of the clinical picture with the lesions is only made post mortem.

In a group of papers published in the first decade of this century, Liepmann established the clinical pictures of apraxic disturbances from lesions in the supramarginal gyrus region and from lesions of the corpus

callosum. Since that time Liepmann's conclusions have been amply confirmed. Even such workers as von Monakow who purported to be highly critical of Liepmann actually confirmed his results. It is of interest that Kurt Goldstein (1908) was one of the first after Liepmann to publish a case of callosal disconnexion with motor disturbances.

Liepmann's analysis of the Regierungsrat was based on a disconnexion approach. Unfortunately the very fact which makes Liepmann's accomplishment so remarkable, namely, that he was able clinically to separate out very precisely the patient's many disturbances into a small group of functional impairments, each correlated to a particular lesion, makes the case a very difficult one from the heuristic view. I will, therefore, use instead as a case illustrative of the mechanisms of apraxia the patient reported by Edith Kaplan and myself (Geschwind and Kaplan, 1962), the mechanism of whose disturbance is much simpler. The reader is again referred to the original paper for detailed discussion of the findings.

Our investigation of this case followed on Mrs. Kaplan's discovery that the patient could write correctly with the right hand (despite the presence of a severe grasp reflex and mild pyramidal disturbance in this hand) but not with the left hand which showed no elementary motor disturbance. In addition to this disturbance of writing with the left hand, we found that the patient consistently carried out verbal commands with the right hand but frequently failed to carry them out with the left hand. In classical terms the patient showed an 'apraxia of the minor hand'. By further testing we were able to go beyond this bare statement and found it possible to delineate the mechanisms of these disturbances.

The disturbance of writing with the left hand could be shown to be aphasic. Thus, when the patient wrote to dictation with the left hand he did sometimes produce an illegible scrawl. On many other attempts, however, he would produce perfectly written but incorrect words (e.g. 'run' for 'go', 'yonti' for 'yesterday'); furthermore he misspelled words when typing with his left index finger and could not correct the errors which he was, in fact, able to recognize. By contrast he could copy correctly with the left hand, but he could not with this hand turn print into script although he could copy both print and script as such. This series of studies excluded any elementary 'motor' disturbance as being at the root of the left-sided agraphia.

He often failed, as we have noted, in carrying out verbal commands with

the left hand. He could, however, copy the movement if it was made before him by the examiner. In addition, he would, given an object, handle it correctly with the left hand although he had failed to show how the object would be used in response to a verbal command alone. Thus, 'Show me how to use a hammer' resulted in a failure of movement or an incorrect movement; he could, however, imitate the examiner's movements or could use the actual hammer correctly. With his right hand he showed none of these difficulties.

We interpreted these disturbances as the effects of disconnecting the right motor cortex from the speech area. The post-mortem showed, as I have already noted earlier, an extensive infarction of the corpus callosum which must be regarded as the cause of these symptoms. The aphasic writing with the left hand and the failure to carry out verbal commands with the left hand could quite easily be explained as a result of disconnexion of the right motor cortex from the speech area. The ability to copy writing correctly but slavishly with the left hand; the ability to imitate seen movements with the left hand; the capacity to handle objects correctly with the left hand, all illustrate that the right hemisphere could perform correctly when no information was required from the opposite side. By contrast the inability to 'copy' print into script is an expected one since such a transformation implies the participation of the speech areas. Certain movements in response to verbal command which involved *both* the right and left sides simultaneously were preserved as were facial movements to command. I will deal subsequently with the analysis of these two special classes of movements.

The case has several features worthy of comment. It illustrates that the designation 'apraxic' is an inadequate one unless the stimulus conditions are specified. The left hand in this patient was apraxic to verbal command but not on imitation or object handling; the *right* hand failed to perform correctly when a response was demanded from this hand to somesthetic stimuli applied to the *left* hand. Rather than use the term 'apraxia' it is therefore preferable to specify the stimulus-response combinations which fail.

It is not possible to assert that the failures of this patient resulted from a general conceptual disturbance or a failure of abstract attitude. The patient could not, indeed, pretend to perform an act with the left hand, but he could pretend with the right hand! It would seem highly unlikely

that a disturbance of conceptualization or of the abstract attitude could be confined to one-half of the body. 'Dementia' as an explanation runs up against the same difficulties. Hysteria or malingering cannot, of course, be seriously considered at all.

The case also points to the probable incorrectness of the classical distinction between aphasic and apraxic agraphia. Many writers have assumed that agraphia confined to the left hand must be 'apraxic'. According to this argument an apraxic agraphia is the result of failure to form the individual letters properly and should result in a pattern of meaningless loops and scrawls. Further support for the distinction between aphasic and apraxic agraphia came from the argument that aphasia could not be confined to one limb. Our case, however, showed both 'apraxic' and 'aphasic' types of writing defect in the left hand. In the early stages his writing was in fact usually but not invariably a totally illegible scrawl. But, particularly after some improvement had occurred, he produced perfectly written words which were incorrect, e.g. 'yonti' for 'yesterday'; his failures at typing were, of course, also misspellings. It would not be possible to term these other than aphasic errors. It seems unnecessary to assume the presence of two separate writing disturbances in the patient. Both types of error (i.e. incorrectly formed letters and aphasic words) can easily be seen as resulting from a disconnexion between the speech area and the motor cortex.

I have also made no attempt to deal with this disability in terms of the classical grouping of apraxias into limb-kinetic, ideomotor, and ideational which Liepmann (1905a) advanced. It would lead us too far astray to discuss this classification in any detail in this paper. It is my opinion that this classification has not been useful and that it has in fact frequently proved very confusing. It is interesting that the classification plays almost no role in Liepmann's earlier classic discussions of the Regierungsrat and even in the later writings is clearly secondary to the intimate discussion of mechanisms. This formal listing of types of apraxia probably had in practice the unfortunate effect of overshadowing Liepmann's much more important detailed analyses of the mechanisms underlying disturbances in motor performances.

The occurrence of such unilateral agraphias is also of considerable interest when the hand involved by the agraphia has been the one generally used by the patient for writing. Thus, Nielsen (1946) described the

remarkable case of a man who had been taught to write with his right hand but used his left hand for all other skilled movements. He suffered a transient left hemiplegia; this was accompanied by loss of speech, strongly suggesting that the right hemisphere was dominant for language. Some months after recovery from this episode he developed a paralysis of the right leg and lost the ability to write with his right hand. He could, however, write with his left hand although he had not used the left hand previously for this activity. In interpreting this case one must assume that although the patient had always written with the right hand, the left hemisphere had, in fact, always been passively under the control of the speech area in the right hemisphere.

This case is also useful as a comment on an old idea, that cerebral speech dominance is much weaker in illiterates and that the act of writing, being unilaterally performed, influences the opposite hemisphere (*see* Critchley, 1962, for a brief review of the history of this idea). Cases like the one just cited, however, cast serious doubt on this possibility since it is clear that Nielsen's patient had not succeeded in establishing speech in the left hemisphere. The case also makes somewhat unlikely the old suggestion that training in ambidexterity makes a severe aphasia less probable. This, of course, is not in conflict with the assertion that those who are naturally ambidextrous have milder aphasias (Zangwill, 1960).

2. *Extension of the Theory of the Apraxias*

In the preceding section I have developed the theory of the simplest type of apraxia, that due to disconnexion of motor regions from the speech area and from other sensory inputs. The interpretation of the case of Geschwind and Kaplan is clear: callosal disconnexion in man prevents the right hemisphere from carrying out language tasks and also from performing those tasks for which the stimulus comes from non-language sensory areas of the left hemisphere. This theory depends on the assumption that the right hemisphere will deal with non-language tasks correctly as long as appreciation of the stimulus and production of the response both depend on the right hemisphere itself. The patient of Geschwind and Kaplan exhibited independence of the right hemisphere in non-verbal tasks; this hemisphere functioned successfully in imitation of movements, object handling and slavish copying.

A deeper search into the literature and further clinical observations soon

convinced me that the independence of the right hemisphere in non-language functions which was manifested by our patient is not universal and may indeed be the exception. It was the work of Liepmann which again contains the earliest and probably the best evidence for this view.

The first description of the clinical picture of extensive disconnexion of the corpus callosum is contained in the paper of Liepmann and Maas (1907). Their patient (Ochs) had a right hemiplegia which at post-mortem was found to be due to a lesion of the pyramidal tract in the left pons. The patient could carry out many actions very well with his left hand, e.g. buttoning his coat if the hand was placed on the first button, or carrying a glass of water (if it was placed in his left hand), to his lips. He failed to write with the left hand; in fact, he could not even copy. When given anagram letters he could not form his name with his left hand; instead he collected all the *m*'s (yet when asked, he could correctly say the letters of his name). He made many errors in carrying out verbal commands with the left hand but selected the correct response from a series of movements made by the examiner. He did not, however, improve on imitation. This patient frequently mishandled objects placed in the left hand. At post-mortem there were found the already mentioned lesion in the pyramidal tract in the pons as well as an infarct of the corpus callosum which spared the splenium. Other patients with callosal lesions have shown a similar disturbance to that shown by Ochs. Not only can they not carry out verbal commands with the left hand, write with this hand, or form words with anagram letters (in which respects they resemble the case of Geschwind and Kaplan), they also show significant impairment in imitating seen movements with the left hand and difficulties in object handling with this hand. The case of Bonhoeffer (1914), for example, showed these deficits. Why do the cases fall into two such groups? It is interesting that Maas (1907) himself called attention to this problem. His explanation was that it depended on the intensity of the callosal lesion, but I do not believe that this is correct.

Because the first case of 'motor' disturbances due to callosal disconnexion seen by Liepmann and Maas showed the more extensive syndrome of left-sided apraxia to verbal command, on imitation and in the handling of objects, these authors regarded this clinical picture as the 'normal' syndrome of callosal disconnexion. Liepmann explained the data by assuming that the left hemisphere is dominant not only for speech but also

for movement. This dominance was assumed to manifest itself trivially in the usual greater dexterity of the right hand; a more important aspect of this dominance, however, was that the left hemisphere contained the 'memories' of movements.

Evidence from other types of patients support Liepmann's contentions that the left hemisphere is dominant for more than speech. Thus, Liepmann (1905b) described the syndrome of apraxia of the left hand in patients with right hemiplegia and aphasia ('sympathetic' dyspraxia as it is sometimes called). I have reinvestigated this problem (Geschwind, 1963a) and been able to confirm Liepmann's observations in detail. This syndrome is of special interest both because of its great frequency (it is, in fact, much the most common cause of apraxic disturbances) and its theoretical interest. Liepmann found this syndrome in 14 of a group of 18 patients with right hemiplegia and severe aphasia. By contrast it occurred in only 6 out of 23 right hemiplegics without severe aphasia. It did not occur at all in the right hands of a group of left hemiplegics nor in a group of senile and demented patients. These controls effectively remove the possibility that the syndrome is the non-specific result of brain damage or dementia. They also show its close relationship to lesions producing aphasia. The fact that it did not appear in all the hemiplegic aphasics and that it appears occasionally without aphasia suggests that the relationship is one of anatomical propinquity of lesions; the apraxia frequently accompanies the aphasia but is independent of it.

The syndrome of sympathetic dyspraxia is essentially the same as that described by Liepmann as occurring in the left hand in callosal lesions. The patient carries out verbal commands either very poorly or not at all with the left arm. Demonstration by the examiner helps in some instances but in most cases little or not all at. Usually the actual handling of objects in the left hand is significantly better than movements to command or in imitation of the examiner, but major failures in the handling of objects do occur in a significant minority of these patients.

Several interpretations of this syndrome can be excluded. I have already noted the impossibility of attributing this picture nonspecifically to brain damage or dementia. One obvious interpretation is that since these patients are generally aphasic, their failures to respond to verbal command are the result of comprehension deficit. Liepmann showed in several ways that this interpretation is untenable. In my cases I have also been abie to

rule this out. Thus, one of my patients showed this syndrome in especially clear form, failing very badly on verbal commands and imitation and also doing poorly in object handling. He had a marked limitation of speech, being able to produce only single words and never producing a sentence. He could, however, answer questions very well with single words. He failed to carry out very simple commands such as to point to the floor but correctly answered verbally much more complex questions as "What occupation were you engaged in before you became ill?" and "Can you name some tools used by carpenters?" Furthermore, the patient could answer specific questions about the task. When asked, "Do you know how to use a hammer?" he said, 'Nails' but could not show how a hammer was used. He could, however, indicate when the examiner had made the correct movement although he could not produce it himself. In other cases a strong clue that comprehension is intact is given by the fact that the patient may respond with a movement that is a clear distortion of the correct response. For example, a patient exhibiting this syndrome when asked to salute may place his hand in the proper attitude but several inches in front of his forehead. These facts all contribute to rule out incomprehension as the cause of the failures. They also highlight a very important aspect of the examination of aphasia. Many textbooks suggest examining for comprehension by means of the ability to carry out verbal commands. It is obvious from the results I have cited that while the ability to carry out verbal commands indicates retained comprehension, its absence does not exclude the preservation of excellent comprehension.

The fact that my patient could give good single word verbal responses while failing to carry out verbal commands probably rules out another possible interpretation. Some would argue that aphasia is not a disturbance of speech but one of symbolization and that my patient was incapable of symbolic manifestations in any form. The patient would under this interpretation be regarded as having lost gesture as well as spoken language. However, aside from the difficulty of regarding some of the movements involved as symbolic, it would be necessary to assume in my patient that *non-verbal* symbolization was worse than *verbal* symbolization! Once one admits the separability of the disturbance in movement from the disturbance in language, the concept of a general asymbolia loses its value.

Liepmann felt that this group of patients consisted predominantly of

cases with lesions near Broca's area as evidenced by their aphasic symptomatology (paucity of speech with good comprehension). He thought that the lesions probably would be found in the white matter immediately beneath Broca's area but above the internal capsule. A lesion in this location would involve not only descending fibres but would also destroy fibres going into the corpus callosum and thus to the opposite side. In those brains which he had had the opportunity to observe at post-mortem he did find such subcortical lesions. He did not make clear exactly from what part of the cortex of the left frontal lobe the callosal fibres originated, a point of importance to which I will return. It is clear that he thought the lesion resulted in disconnexion of the right motor region from some portion of the left frontal region; the lesion was regarded by Liepmann as cutting off the right hemisphere from the memories of movements stored on the left side.

3. *The Apraxias of the Supramarginal Gyrus Region*

Further details of the above mechanism can be still further elucidated by considering another group of 'apraxic' patients, those with lesions of the left supramarginal gyrus region. Liepmann (1900, 1906) first described this syndrome in the famous case of the Regierungsrat. The general picture which emerges from the literature is a consistent one. Unilateral lesions penetrating deeply in this region may lead to an apraxia which is either equal bilaterally or worse in the right hand. Except for its bilaterality, it is similar in its pattern of disabilities to that usually seen in the two preceding conditions (apraxia from a callosal lesion and 'sympathetic dyspraxia'). The patient performs badly to verbal command. imitation of the examiner is usually poor and object handling is involved, but less often. Liepmann stressed that the responsible lesion was one which cut off white matter connexions (lying deep to the supramarginal gyrus) between posterior parts of the left hemisphere and motor regions. The retained connexions on the right side would not have been able to substitute, in most cases, since there had been no learning on the right side. The lesion in the supramarginal gyrus would cut off connexions running from Wernicke's area to motor regions via the arcuate (superior longitudinal) fasciculus and also connexions from the visual region to the motor area.

There is a problem raised by this simple disconnexion hypothesis. This lesion deep to the supramarginal gyrus should disconnect Wernicke's area

from motor regions and the visual region from motor regions; the failure of the patient to carry out movements to verbal command or on imitation is therefore understandable. Why should movements be impaired in the actual handling of objects? There are several possible explanations, but we cannot be sure which, or indeed, whether any of them is the correct one. Liepmann suggested the following mechanism. He asserted that even the manual handling of objects was frequently learned 'visually'. One can cite simple examples which are suggestive. Many acts, such as combing the hair, may be routinely carried out under visual control. When they are done without vision Liepmann's suggestion would be that they are done with 'visual mediation'. In other words cross-modal transfer would be involved in the learning of many tasks, and the performance under tactual control would depend on transfer from visual learning.

Liepmann brought forward as evidence for this view the pattern of *retained* responses to somesthetic stimulation. He pointed out both for the Regierungsrat and Ochs that while they handled most objects poorly, they performed well on somesthetic stimulation in those tasks which were highly overlearned and which were usually done independently of vision. Thus, the Regierungsrat and Ochs both failed in some very simple object manipulations, yet were able to button their clothing blindfolded so long as the hand was first passively placed on the garment. Liepmann attributed this to the ability of the isolated sensory and motor cortex to do this highly overlearned task without the mediation of vision. Liepmann made use of the preservation of such movements as important evidence. It enabled him clinically to rule out any sort of elementary motor or sensory disturbance since these would be incompatible with the deft buttoning of a coat while blindfolded. This fact emphasizes again the point that 'apraxia' is not a unitary disturbance since under appropriate conditions these patients could carry out complex motor tasks.

There is another possible explanation for the difficulty in object handling. Liepmann had felt it necessary to account for the fact that the patients could not carry out all tasks of object handling correctly even though the primary sensory and motor cortex retained their direct connexions. We know that there are connexions via U-fibres between the postcentral and precentral gyri. However, are these connexions used for the learning of complex tasks under somesthetic control? It is not at all unreasonable to assume that for the learning of such complex tasks the pathway from

primary sensory to primary motor cortex may be via association cortex, as in the case of connexions of other modalities to the motor system. The pathway from sensory to motor cortex would thus run from sensory cortex via somesthetic association areas and then would travel forward to the motor region. If this were the path then a lower parietal lesion might well involve part of this somesthetic-motor pathway. Sparing of this pathway in its entirety might leave object handling totally unaffected. If this explanation were correct then one would have to assume that the preservation of some movements depended on their having been overlearned, i.e. on their being so redundantly represented in the somesthetic association cortex that partial lesions left them unimpaired. This problem of the factors affecting movements made under somesthetic control is one which obviously deserves further study.

Liepmann repeatedly insisted that the critical lesion in the region of the supramarginal gyrus involved not the cortex but the white matter running beneath the gyrus. An alternative explanation would be that the 'memories for movements' were in the supramarginal gyrus region, i.e. that the cortex was involved. Against this and in favour of the notion of white matter disconnexion is the fact that lesions *anterior* to the left Rolandic fissure produce apraxia on the left side of the body despite an intact left supramarginal gyrus. One would expect that, if the *cortex* of the left supramarginal gyrus were the important structure and were not involved by the lesion, the impulse could travel across the callosum to the right supramarginal gyrus and thence forward to the right motor region. In this instance no apraxia should be seen. The other possible pathway for control of the left side of the body involves a path running beneath the left supramarginal gyrus to the left frontal lobe and from here to the right hemisphere. In this case apraxia of the left side could occur with a lesion anterior to the left supramarginal gyrus.

The sinistral unilaterality of the pathway in most people for the carrying out of acts by the motor cortex under sensory stimulation is emphasized by the rarity of well-attested cases of left-sided apraxia occurring as the result of an isolated right parietal lesion. Foix (1916) emphasized right parietal lesions as a cause of left-sided apraxia, but his evidence is not convincing. He even said that 'Liepmann's principal case' had a right parietal lesion. but since this statement applies neither to the brain of the Regierungsrat nor that of Ochs, we may dismiss this assertion as simply being

incorrect. Hécaen and Gimeno Alava (1960) have discussed this problem. They found 16 cases, 3 of their own, the remainder from the literature, in which patients with apraxia of the left side had clinical evidence of a right hemispheric lesion. However of these, 11 almost certainly also had lesions of the left hemisphere. Among the remaining 5,2 were known left-handers. Of the remaining 3,1 came to post-mortem and showed no involvement of the left hemisphere. If one can accept these findings it would appear that a right parietal lesion causes apraxia of the left hand only exceptionally unless there is an associated left hemispheric lesion. This conclusinn is further borne out by the study of Ajuriaguerra *et al.* (1960). If we consider only those cases in their series which are relevant to our discussion, we find that they had 58 cases of apraxia (divided by them into 11 cases of ideational apraxia and 47 cases of ideomotor apraxia), of which 48 occurred in their 206 cases of left-sided post-Rolandic lesions, and 10 occurred in their 55 cases of bilateral lesions. There was not a single case of apraxia in their 151 cases of right-sided post-Rolandic lesions.[22] I would in fact expect that there exist occasional cases in which a right supramarginal gyrus lesion might lead to an apraxia of the left arm on imitation and object handling (but possibly not in response to verbal command). This unusual situation would exist only in those patients in whom the right hemisphere was relatively independent.

I have already noted that deep to the supramarginal gyrus are fibres from the visual association areas running into the frontal lobe and fibres from the speech area (which is, of course, auditory association cortex) also coursing to the frontal lobe. These are probably the pathways by which motor responses are carried out in response to complex visual or auditory stimulation. Where in the left frontal lobe do they terminate and by what precise pathway does stimulation get across to the opposite motor region?

One's first inclination would be to assume that these pathways terminated in area 4, the primary motor cortex. But again, the rule of Flechsig (1901) comes into play. The motor cortex, at least in the primate, receives no direct fibres from the visual association cortex. Chusid *et al.* (1948) produced a most instructive study of the frontal connexions of the visual association cortex in the macaque which brought out clearly the fact that many of these important connexions arise from the tissue buried in the depths of the lunate sulcus of the occipital lobe. They found with strychnine neuronography that the anterior bank of the lunate sulcus

strongly fired the cortex immediately anterior to itself. The only distant region where they found strong firing was the cortex of both banks of the arcuate sulcus of the frontal lobe and the cortex immediately anterior and posterior to this sulcus. There was no firing in the motor cortex proper. The posterior bank of the arcuate sulcus in turn fired strongly into areas lying immediately posterior to itself including the *homolateral* area 4 and also strongly fired the *contralateral* arcuate sulcus region. Bonin and Bailey (1947) report that FA (area 4) received fibres from FB (lying above the superior half of the arcuate sulcus, often called area 6) and from several areas in the parietal lobe but not from any other part of the frontal lobe. Furthermore, FA had no callosal connexions in the primate except for some restricted parts of the trunk and lower face regions. Their results and those of Chusid, Sugar, and French thus are complementary. Similarly, Bailey, Bonin, and McCulloch (1950) state that in the chimpanzee the most numerous afferents to FA come from FB and PC (the postcentral gyrus). The pattern of callosal projection is the same as in the macaque.

Krieg (1954) studied the efferent connexions of the frontal lobe in monkeys by the Marchi method. He found that each area 6 has strong callosal connexions to the opposite area 6. In addition each portion of area 6 sends numerous fibres to the part of area 4 directly behind it. Krieg's findings are therefore in keeping with the physiological findings of the workers cited above. Krieg did find callosal fibres from area 4. He comments, however, that the number and calibre of these fibres is very small and his illustrations of the actual lesions and the consequent degeneration support this assertion. Krieg notes that his anatomical findings are in keeping with the poor callosal responses obtained from area 4 by Bailey *et al.* (1950).

We might guess from these data that connexions between visual and motor cortex run as follows: from the visual cortex proper to the visual association cortex to area FB (roughly area 6) and thence to the motor cortex. Area FB thus becomes the association cortex of the motor system. Similarly, the callosal connexions of the motor system would have to be via area FB.

The view that the premotor cortex acts as the association area of the motor cortex receives support from the connexions to the motor region of the auditory system. Thus, Sugar *et al.*'s (1948) study showed that the important frontal projections from the supratemporal plane are to the

cortex lying within the depths of the arcuate sulcus and to the cortex immediately posterior to this sulcus, in particular to the regions behind the lower limb of the sulcus. In other terms the projections from the supratemporal region are to areas 8, 6b (lower half of area 6), and 44; there are no direct connexions from the supratemporal plane to area 4. The earlier study of Ward *et al.* (1946) which was the first to discover afferent connexions to area 6 had also revealed the connexions from the supratemporal plane to this part of the frontal lobe.

Thus, it would appear that the auditory system like the visual system projects to this 'motor association cortex' lying anterior to area 4, and that this region in turn projects to area 4. It seems reasonable that this is the pathway by which motor tasks are carried out to auditory stimulation.

These studies all support the theory that connexions from primary sensory areas to the motor cortex involve a multisynaptic pathway which travels by way of the association areas adjacent to the sensory areas in question and the 'motor association areas' lying anterior to the motor cortex proper. The startling experiments of Welch and Stuteville (1958) are readily explained by this theory and also constitute further evidence for it. These authors placed small lesions in the depths of the posterior part of the superior limb of the arcuate sulcus in monkeys. Confirming earlier results by Kennard and her co-workers (summarized in Kennard, 1939) they found that these animals disregarded visual stimuli on the side opposite the lesion, and indeed even collided with objects on that side. The animals did not respond to auditory stimuli coming from the side contralateral to the lesion by turning towards the stimulus (as a normal monkey would); instead they turned towards the side opposite the stimulus. Finally. these animals exhibited little response to tactile or painful stimuli on the side opposite the lesion. These animals showed a marked poverty of movement on the involved side. Tactile and visual placing reactions were lost on that side. Recovery from this dramatic syndrome began in five to seven days and was complete in two weeks.

Welch and Stuteville state, "How vision, hearing, tactile sensibility, movement and placing reactions are so seriously impaired by a lesion of the cortex which does not directly interfere with any of the several receiving areas or the motor area of the cortex is difficult to understand." The results, however, become easy to understand when one considers that the

area involved by the lesion is in fact part of the pathway from all sensory modalities in one hemisphere to the motor cortex of that hemisphere. The lesion therefore effectively disconnects the motor cortex of that side from all sensory stimuli. Why does the animal not respond with the hand opposite the *normal* hemisphere to stimulation on the side opposite the damaged hemisphere? I would suspect that it is probably because the lesion has also cut off the origin of callosal fibres between the two areas 6. This explanation of the Welch and Stuteville data may well apply to other studies in which lateral frontal lesions have been said to lead to difficulties in sensory discrimination (e.g. Weiskrantz and Mishkin, 1958) – the real deficit may be in motor response.

Some aspects of the Welch and Stuteville experiments deserve brief comment. Ettlinger and Kalsbeck (1962) found a loss of tactile placing responses on the side contralateral to either a lesion of the primary somesthetic cortex or of the parietal association areas. Welch and Stuteville found that these responses disappeared in their experiments. The normal pathway for a tactile placing reaction would appear to travel not via the U-fibres connecting the primary somesthetic and primary motor cortex but rather by way of the association areas. A striking finding of the Welch and Stuteville experiments was the transience of this dramatic disturbance. This is another example of the tendency for small association area lesions to be compensated. Was the compensation by way of normal cortex adjacent to the damaged region, or were the animals using totally new pathways? This remains to be studied. There was an even more startling result in the Welch and Stuteville study. An animal showing this syndrome behaves as if he is blind in the field opposite the lesion. Following a removal of the occipital lobe on the side opposite the lesion there is a return of responsiveness to stimuli in the remaining half of the visual field although the animal continues to neglect tactile and auditory stimuli on that side. It would appear that some alternative pathways for motor response to visual stimulation of the hemisphere on the side of the frontal lesion are kept inhibited as long as the opposite visual region is operative. It would be most interesting to trace the anatomical substrate of this inhibition. If it is mediated via the corpus callosum it would appear likely that callosal section might cause the entire syndrome to vanish!

The first case of Hartmann (1907) might appear to be a human example of this syndrome of marked inattention to all modalities of stimulation coming from one side as the result of a frontal lesion. There are, however, certain difficulties in the clinical picture and pathology which prevent the full use of this case. Hartmann, however, must be credited with considerable prescience for he argues that since the right arm of his patient showed no movements in response to sensory stimuli, the frontal lobe (i.e. the region anterior to the motor cortex on the left side) was necessary for the conduction of sensory stimuli to the central gyri. This would agree of course with the thesis presented here that the premotor region is a waystation on the route from sensory cortex to primary motor cortex.

I have postulated a mechanism for the response of the motor cortex to sensory stimulation. If this thesis is correct we must assume that the

apraxia of the left limbs in the patient with a right hemiplegia is not the result of the part of the lesion causing the hemiplegia. Rather it is the result of coincidental damage either to area 6 or to the callosal fibres to which it gives rise. Alternatively it is the result of damage to the afferents passing to area 6 from the visual, auditory, and somesthetic regions and travelling perforce beneath the motor cortex.

There are certain implications of this argument which are worth considering for the moment in more detail. The argument implies that a lesion in man of the regions anterior to the motor cortex on the left, but sparing area 4, should produce bilateral apraxia in our sense, i.e. failure to carry out movements to command and on imitation of the examiner but with variable effects on actual object handling. As many readers will know it has in fact been asserted for a long time that lesions anterior to the motor cortex lead to apraxic disturbances (*see*, for example, Nielsen, 1946; Aring, 1944) and it has often been argued that these are 'limb-kinetic' apraxias. Perhaps the best known of these disturbances from lesions anterior to the motor cortex are the facial apraxias (whose discussion I leave to the next section) and the pure agraphias which are said to arise from the posterior end of the second frontal convolution. I will not discuss the problem of 'frontal apraxia' in detail since my own acquaintance with this large literature has not been intensive. My feeling has been that 'limb-kinetic' apraxia has not been defined clearly enough to separate it from mild pyramidal disturbance. Liepmann (1905a) himself must have been somewhat unsure of limb-kinetic apraxia since he used as an example of this type of patient no case of his own but rather one published many years earlier by Westphal. The apraxias of the left side accompanying Broca's aphasia, which I have already mentioned, are due either to lesions of area 6 itself or of the callosal fibres arising from it. There is no clear-cut evidence to show that a lesion of the left-sided area 6 itself would produce a bilateral apraxia in virtue of its cutting off connexions both to the homolateral area 4 and to the contralateral motor region. The reason that such cases are lacking is probably the result of the fact that lesions of the left area 6 are very likely to encroach on the left area 4 and therefore to produce so much weakness in the right hand as to make assessment of apraxia difficult.

I believe, however, that this is an area where animal experimentation may be of greater use than it has been. Earlier experiments on ablation of

area 6 have generally stressed 'motor' functions (*see*, for example, the discussions in Bucy, 1944), rather than learning activities. The work of such investigators as Kennard (1939) Welch and Stuteville (1958), Weiskrantz and Mishkin (1958) should be extended. Of parallel interest would be further knowledge of way stations to the hippocampal region from area 6.

4. *Facial Apraxia*

This term is used as a shorthand for 'apraxia of the cranial musculature', i.e. inability to carry out movements of this musculature either to command or on visual or tactile stimulation. It is the first form of apraxia to have been recognized (although not under that name) by Hughlings Jackson (1878) who commented on the inability of certain aphasics to protrude the tongue. It has continued to arouse interest sporadically. Many authors would, in fact, like to include Broca's aphasia as a *part* of such an apraxia.

Certainly this disturbance is the most common apraxic disturbance in aphasics. It has probably been recognized much more often than the more extensive apraxia of the left side which I have mentioned earlier. I suspect that one reason for this is that when the patient fails to carry out facial commands but does perform limb commands, it is clear that comprehension is intact. When all commands on the intact left side are not performed, it is all too easy to attribute the failure to incomprehension.

The clinical picture is essentially similar to that of other apraxic disturbances but has some distinctive and intriguing characteristics. The patient usually does most poorly in carrying out facial movements to verbal command. He may simply fail to perform at all or may make an incorrect movement, e.g. he may open the mouth when asked to protrude the tongue, or blow instead of suck. He may make movements with one of his limbs to carry out the demanded task; thus, he may pretend to stub a match in an ash-tray or stamp on it with his feet when asked how he would blow out a match. He may remove imaginary crumbs from his lips with his fingers and not with his tongue even when asked repeatedly to use his tongue; he may even insist that he has always performed this action with his hand. Most interesting of all, the patient may echo the command or produce onomatopoietic responses. Thus, when asked to cough, he may say, 'Cough'; when asked to blow out a match he may say, 'Blow' or even, 'Blow out a match', or 'Puff'. This type of response is all the more striking

in that these verbalizations or vocalizations do not appear when the patient carries out the command correctly; similar disturbances are much rarer when patients fail to carry out *limb* movements to command.

The special peculiarity of facial movements is, of course, that they are generally bilateral. We must therefore ask how this bilaterality is achieved. A common view is that facial movements are integrated via the corpus callosum. This seems somewhat reasonable in view of the assertion that by strychnine neuronography (McCulloch and Garol, 1941) there can be demonstrated callosal connexions only between very limited parts of area 4, comprising the trunk and neck divisions and part of the face division. There is some reason, however, to question whether in man even these callosal connexions are of importance. Thus, the patient of Geschwind and Kaplan (1962) who performed poorly in carrying out commands with the left hand or left leg moved his face *bilaterally* in carrying out facial commands. This suggests that the pathway by which facial commands are carried out descends unilaterally to some brain-stem level where bilateral facial movement is integrated. It may be argued that a unilateral lesion of the face area of the right cortex produces weakness of the left lower face. This weakness, however, may be the effect of the withdrawal of tonic influence resulting from destruction of the right cortical face area and is probably not good evidence that movement of the left face to verbal command is mediated via impulses passing over the callosum. The right cortical face area would thus be regarded as providing a constant tonic influence but as having a phasic effect only under special conditions. Alternatively one might speculate that normally these bilateral facial movements are integrated via the callosum but that when the callosum is gone there is ready replacement by means of directly descending pathways providing bilateral innervation. I think that this explanation is less likely than my earlier one, i.e. that each cortical face area separately is normally capable of causing bilateral activity at a brain-stem level.

We can now consider the pathway for facial movements to command or on visual stimulation. The pathway for facial movements to verbal command probably goes from the posterior speech area via the arcuate (superior longitudinal) fasciculus to the association cortex lying anterior to the face area. Similarly, the pathway to the face area from the visual region probably also passes in the inferior parietal region to some area anterior to the Rolandic face area.

The first implication of this is that lesions of supramarginal gyrus region should lead to facial apraxia. It has been our experience that they commonly do so. In particular facial apraxia to command and visual stimulation is likely, in our experience, to accompany the clinical syndrome of conduction aphasia, whose importance and whose anatomical basis have been emphasized lately by Konorski, Kozniewska and Stepien (1961) and to which I will return in a later section. These patients show no hemiplegia in the great majority of cases. Their speech may be fluent with many phonemic paraphasias. Even when speech is limited there are runs of fluency. While mild dysarthria is occasionally seen, the great effort and marked dysarthria of the Broca's aphasic are absent. Despite good to perfect comprehension there is marked difficulty in repetition. Their lesions probably lie low in the arcuate fasciculus (Konorski *et al.*, 1961). It is my opinion that the accompanying facial apraxia results from the same lesion. The fact that limb apraxia may be absent suggests that the fibres intended for facial 'association cortex' run lower down than those going to the motor association cortex for the limbs.

The common occurrence of facial apraxia with lesions near Broca's area is readily understood. The lesion may destroy the association cortex anterior to the face area. Facial movements cannot be carried out to command or on visual stimulation because the lesion has cut off connexions to the left face area and cut off the origin of callosal fibres to the right face area. Alternatively a lesion in this neighbourhood may destroy left cortical face area or its projection fibres. In this case facial movements to command or visual stimulation must be carried out via the right face area receiving stimulation over the corpus callosum. If the lesion extends deep into the white matter it can involve the callosal fibres and facial apraxia will result. When a tactile stimulus. e.g. a drinking straw in the mouth, is used, the act, if overlearned, is often carried out correctly via the right sensory and motor cortex. In brief, we are regarding the facial apraxia of the patient with a Broca's aphasia as part of the left-sided apraxia of these patients. Facial apraxia is more common than apraxia of the left limbs in this group of aphasics because a lesion producing aphasia will usually destroy the association areas and callosal fibres involved in face movements but will often spare those associative connexions lying more superiorly which are involved in limb movements.

It the association cortex involved in facial movements to command or

visual stimulation the same as Broca's area? I am not certain but some evidence suggests that they are different. For one thing, although some authors have chosen to regard Broca's aphasia as an apraxia of the speech organs, it has been clear in my experience that a very severe Broca's aphasia may be accompanied by little or no apraxia of cranial musculature; thus, some patients carry out movements with the cranial musculature to command and may imitate well despite marked restriction of speech. This would suggest some difference in the anatomical arrangements for speech and other cranial movements. Furthermore, I have already noted that patients with conduction aphasia commonly show facial apraxia and yet may show an aphasia which is quite different in character from a Broca's aphasia; in particular, dysarthria may be mild or absent. This too suggests that Broca's aphasia and facial apraxia may vary independently even though facial apraxia is most often seen with Broca's aphasia. Whether these differences are related to differences in the association cortex involved remains an open question. In any case, it is probably unjustifiable to call Broca's aphasia an apraxia if by this is suggested that it comprises part of a picture in which facial apraxia is necessarily present. The frequent tendency of the patient with facial apraxia to *repeat* the command which he fails to carry out also suggests a difference in anatomical arrangements for facial movements and speech. It cannot be ruled out that these differences are the result only of different degrees of overlearning of speech and non-speech movements.

5. *Whole Body Movements*

I will not discuss here the vexing question of what has sometimes been called 'frontal apraxia' of gait and its possible anatomical substrate. I have not seen a clinical picture to which I would with assurance apply the term 'apraxia of gait' although I have seen many examples of frontal gait disturbance. For a gait difficulty to qualify as an 'apraxia of gait', it would have to meet the following criteria: the patient should perform whole body movements poorly to command or on imitation; the errors should not merely be failures to perform or clumsiness but should also include substitution of other well-performed movements for the desired ones. By contrast, under certain conditions, e.g. appropriate somesthetic stimulation, the patient should perform whole body movements perfectly well. This syndrome has not been seen to my knowledge – or at least not

recognized. The patients generally described as manifesting frontal gait disturbances show a fixed disturbance in performing integrated whole body movements, and I would accept Denny-Brown's (1958) view which regards this disturbance as involving impairment of more elementary motor mechanisms of some type. Similarly, I have not seen apraxic disturbances in the limbs in my cases of frontal gait disorder, i.e. my patients have made leg movements correctly to command and on imitation; I can conceive, of course, that in some instances apraxias of the legs might accompany a frontal gait disturbance but it is certainly not an obligatory association.

What I will stress in this section is rather the apparently anomalous *preservation* of whole body movements in patients with otherwise widespread apraxic disturbances and will suggest a mechanism that may underlie this finding. I have now seen several such patients. The only paper in which to my knowledge such sparing is noted is Liepmann's (1900). The patient had failed to carry out limb movements to command and was thought initially to be suffering from a profound comprehension difficulty. However, Liepmann wrote, "Against the view that comprehension of speech was totally lost was the circumstance that the patient promptly carried out tasks which he could execute with the whole body, such as standing up, walking to the window or walking to the door." This brief and incisive observation was thus the first clue that the patient was not so severely demented as he had been regarded for some time. Liepmann did not, however, return to the discussion of this point and did not attempt to analyse the mechanism of the preservation of whole body movements.

It seems clear that the callosum is not necessary for the integration of this type of bodily movement. The patient of Geschwind and Kaplan (1962) who often failed to carry out movements to command with the individual limbs of the left side still walked normally to command. It seems likely that the act of walking is controlled as an integrated act at the level of the brain-stem and that a command may descend unilaterally to this integrating system and set it going.

In another patient whom I have investigated, there was a left parietal glioblastoma. The patient showed no significant motor findings but manifested a marked bilateral apraxia of face and limbs. He performed very poorly to command, showed little or no improvement in attempting

to copy movements made by the examiner and even handled objects very poorly; e.g. he could not with either hand hold a hammer properly to extract a nail although this particular manoeuvre was demonstrated to him several times. In striking contrast was his preserved ability to carry out whole body movements. He could carry out a command such as, "Stand up, turn around twice and then sit down again," although he had been unable to perform such simple commands as, "Make a fist." Even when he failed to execute a movement, e.g. a bow, correctly to command, he immediately carried it out after the examiner had demonstrated the movement. There was no clumsiness at all and indeed it was generally accepted that the patient's bow was considerably more graceful than that of the examiner.

One of the most dramatic manifestations of this discrepancy was seen when the patient was asked to assume the position of a boxer. He immediately assumed the boxing stance, leading correctly with the left fist. When asked to punch he looked perplexedly at his fist. Several different terms were then used – 'punch', 'jab', 'uppercut', but none of these succeeded in eliciting a response. This situation set sharply in relief his ability to perform whole body movements in the face of marked difficulty with movements of individual limbs. I have seen another patient who showed a marked bilateral apraxia, worse on the left side, which involved the face and individual limbs. This case also demonstrated a striking relative preservation of whole body movements.

These cases have several points of interest. The grace and elegance of some of the whole body movements in such patients rules out that their failures in individual limb movements are the result of some general clumsiness or inco-ordination. The preservation of whole body movements cannot be interpreted as resulting from the greater simplicity of the movements. It is difficult to see how, "Stand up, turn around twice and then sit down again" is simpler than "Make a fist" but our second patient could do the former and not the latter. Nor is it obvious that "Show me the position of a boxer" is simpler than "Show me how a soldier salutes" (all of these patients were ex-Servicemen). Finally these observations also rule out the notion that apraxia of individual limbs need lead to inco-ordination of whole body movements.

It seems to me that such a marked difference in performance of whole body movements to command must depend on the utilization of different

anatomic arrangements than those involved in movements of individual limbs. The bundle of Türck may well represent the efferent pathway for whole body commands. In the macaque this bundle arises mostly from the posterior part of the superior temporal gyrus (Brodal, in Jansen and Brodal, 1954), the region corresponding roughly to Wernicke's area in man. According to Brodal this tract is present in man and is extensive. All authors who have described it feel that it arises from the posterior temporal region although the posterior parts of all three temporal gyri have been implicated by different observers.[23]

In the macaque, according to Brodal, Türck's bundle descends via the retro-lenticular portion of the internal capsule and the lateral part of the cerebral peduncle to the dorsolateral pontine nuclei. These nuclei project in turn bilaterally to the vermis of the cerebellum. The *fronto*-pontine projections descend to the medial pontine nuclei which also project bilaterally to the cerebellar vermis. Türck's bundle would thus appear to project to a system which on good grounds is regarded as being involved in the motor control of gait and whole body movements. It is of interest that the pontine nuclei receiving afferents from the parietal and occipital lobes do not project to the vermis; their projections go only to the cerebellar hemisphere of the opposite side, in contrast to the bilateral projections of the nuclei receiving connexions from the frontal and temporal lobes. Flechsig (1901) surmised, and I would suspect correctly, that the bundle of Türck subserved "motions of the body and head in consequence of auditory impressions." Further evidence for Flechsig's supposition is Foerster's finding (cited by Crosby *et al.*, 1962) of adversive movements on stimulation of area 22 in man and the occurrence of adversive seizures with lesions in this location. It appears likely that these effects are the result of excitation of connexions from this region travelling by Türck's bundle. I would argue that it is highly likely that the carrying out of whole body postures to command depends on this bundle. The projection from Wernicke's area via this structure could still be intact even when the connexions from Wernicke's area to the motor association cortex were cut off. I would suspect that an appropriate lesion of Türck's bundle might lead to that condition of 'apraxia of gait' in the sense in which I used the term at the start of this section. In the monkey bilateral lesions of this bundle might lead to failure to perform learned whole body movements to auditory stimuli.

6. *Other Bilateral Movements*

We have so far considered two types of bilateral movements, facial movements and whole body movements, and analysed the special circumstances involved in either their special impairment or their preservation in the face of widespread impairment of individual limb movements. We now move to another type of bilateral movement, the type in which individual limbs are involved on both sides. We distinguish two varieties of this type of movement. the bilaterally symmetrical movement (e.g. making circles in the air with both hands), and the asymmetrical learned bilateral movement, e.g. the movements of tying shoelaces.

It is often thought that callosal integration is necessary for the successful performance of such movements. Most of our evidence so far in fact suggests that this is not necessarily the case. Thus, the patient of Geschwind and Kaplan (1962) who often failed to perform actions with the left limbs to command did tie his shoelaces to command using *both* hands correctly. We were perplexed by this at first but then realized that as long as each hemisphere had learned its task such bimanual movements could be carried out. In our case a command to tie the shoelaces would thus have been conveyed to the left hemisphere; the right hand would then move to begin the task. But the visual regions of the *right hemisphere* could then observe the right hand perform; the right hemisphere would thus receive visual stimulation and proceed to do its part of the task. Presumably a more careful analysis of the latencies with which each hand began to do its task would have helped prove this mechanism. The fact that our patient could respond to visual stimulation correctly with either hemisphere supports this interpretation. Our patient carried out other bimanual tasks equally well.

Ettlinger and Morton (1963) have shown preservation of a bimanual task in a monkey after callosal section, and I presume that a similar mechanism is involved.

If this mechanism seems a bit unlikely, it should be recalled that intensely complex co-ordinations of movements can be carried out by totally separate individuals. The members of a corps de ballet succeed in carrying on such well-integrated movements on the basis of visual stimulation and the use of well-learned sequences. Presumably if two separate individuals can carry out such actions, then the two halves of a cerebral hemisphere

separated from each other should be able to do so also, as they apparently did in our patient. Dr. Ira Sherwin (personal communication) has suggested to me that in animals a similar mechanism may be operative, i.e. one hemisphere may learn what has previously been learned by the other, not via the callosum or any other direct nervous connexion but rather by visual observation of the behaviour of the other hemisphere. This notion deserves further experimental investigation.

Presumably such a mechanism for the carrying out of complex bimanual movements in the presence of callosal disconnexion can come into play only when each hemisphere has separately learned its part of the task; therefore, this type of movement should be best preserved in those individuals in whom the right hemisphere has some significant degree of independence in the learning of motor acts, as was the case for the patient of Geschwind and Kaplan. In the older writings, such as those of Liepmann, poor performance of bimanual acts is mentioned as a consequence of disconnecting lesions. My guess would be that in many patients bimanual acts would be poorly performed since the right hemisphere does not in general learn motor patterns as well as the left, as I have noted in my earlier discussion. There will, however, be cases like that of Geschwind and Kaplan where the right hemisphere has learned its task independently.

An alternative possibility might present itself – that bimanual tasks are learned via pathways descending from only one hemisphere and are therefore likely to be preserved, just as whole body movements are. One patient we have observed presents evidence against this supposition. He showed bilateral apraxia, worse in the left hand, to verbal command, on visual stimulation and on object handling. When asked to perform a bimanual act, the right hand could eventually be got with difficulty to perform its role but the left hand could not. In short each hand was as apraxic as it had been in unimanual tasks. Yet, this patient showed a striking preservation of whole body movements. Clearly, the mechanisms of whole body movements and bimanual movements are different.

I would suspect that a similar mechanism explains the preservation of symmetrical but not highly overlearned bimanual movements in patients with callosal disconnexion. Thus when the patient is given the command to make circles in the air, the right hand can begin and the left can then join in. The patient of Geschwind and Kaplan showed this phenomenon.

When his eyes were closed, however, the performance appeared to change somewhat. He then tended to move his arms at different rates, a performance unusual in a normal and then only attained by deliberate effort. We are not certain that we can attribute this lack of synchrony to the absence of callosal connexions since the mild pyramidal signs in the right arm might have affected its functioning in this task. Further observations will be needed in future cases to confirm this result.

7. *The Problem of 'Motor' Versus 'Cognitive' Learning*

I would like briefly to refer here to a problem that I have avoided in the above discussion, i.e. the classical argument in learning theory as to whether motor learning is the formation of 'stimulus-response' connexions or whether it is the learning of a 'cognitive map'. Thus, has the animal who has learned a maze acquired a 'picture' of it or has he learned a sequence of motor responses to a sequence of stimuli? The distinction is potentially an important one for the investigation of disconnexion phenomena. We might consider a hypothetical illustration. Suppose an animal was taught to respond by pressing a lever when a particular visual stimulus appeared and always used, by preference, his right forepaw in making this response during the period of training. If the visual cortex to motor cortex connexions were cut would the performance be lost? If the animal had acquired a cognitive 'map' then the disconnexion between visual and motor association cortex might be unimportant. He might then be able to use pathways descending to the brain-stem and perform the task with a whole body movement, e.g. by sitting on the lever or by making a crude unilateral movement which was subserved by pathways descending directly to the brain-stem. Suppose by contrast the animal is taught to make a series of distinct complex motor movements in response to the visual stimulus and in fact is taught that these and only these will be rewarded. It is much more likely that this learning will depend on the motor cortex and that a lesion causing a disconnexion of the cortical visual and motor areas will permanently impair the performance of this type of task. Another example might be the contrast between learning the pattern of a walking maze and learning to type. The pattern of a maze once learned, a human could get through it readily, walking, crawling, or driving. But knowing the pattern of a typewriter keyboard alone does not solve the problem of typing. Similarly, it will take great effort for an untrained per-

son to play the piano even when he has learned the simple pattern of the arrangement of the notes.

It might appear at first that my analysis here has been exclusively a stimulus-response analysis and hence antagonistic to the idea of cognitive 'maps'. I have actually stressed the simpler stimulus-response stiutations and have not considered such maps. There is no reason, however, for assuming that these two types of 'motor' learning are mutually exclusive. It would be of great interest to study the pattern of lesions in the nervous system of animals which would abolish or prevent the formation of such apparently purely cognitive learning.

Similarly, I have not discussed the problems implicit in the carrying out of such complex commands as, "Draw a star in the pink square." This request is quite different from such commands as, "Show me how you would use a hammer." In the first command we have three separate elements, the auditory command, and the motor response which, however, must be carried out under the control of vision, thus bringing a third system into play. The anatomical pathways for such commands which involve more than two elements represent an intriguing problem for further study.

VI. OTHER APHASIC DISTURBANCES

I have so far not discussed that condition whose name in classical neurology specifically reflected the theoretical assumption that it was the result of disconnexion, i.e. conduction aphasia or Leitungsaphasie. The term, of course, meant aphasia due to failure of conduction, i.e. aphasia due to disconnexion. In this section there will be presented a discussion of this aphasic disturbance as well as of certain other syndromes which are probably the result of disconnexions.

1. *Conduction Aphasia*

Interest in this condition has been revived by the recent work of Konorski and his co-workers (1961). Konorski's presentation at the International Neurological Congress in Rome in 1961 alerted my colleagues and myself to this condition and we have now seen several very characteristic examples. In our experience the clinical picture is the following. The patient usually shows little or no hemiplegia. His spontaneous speech is often, but not always, copious. Dysarthria tends to be absent or mild, and

whatever phrases are produced tend to be fluent. While articulation may be normal, the speech is obviously and often severely aphasic, usually highly circumlocutory and often grossly paraphasic with a tendency particularly to literal paraphasias. There is marked difficulty in naming. Writing suffers along with spontaneous speech. As we have already noted earlier, facial apraxia to command is often marked and may also be present on imitation.

The notable feature of these cases is the marked discrepancy between comprehension and repetition. In the most striking of these cases comprehension is excellent as manifested by the ability to pick out correct and incorrect sentences, and also in many cases by the capacity to carry out verbal commands with the limbs. In striking contrast is the difficulty in repetition. In some instances even the simplest words fail to be repeated. The patient often says, "Say it again" which may give the impression of his not having heard or comprehended; yet even when there is positive evidence of comprehension, repetition does not improve. Thus, a patient may, on being given the word 'president', say, "I know who that is – Kennedy," but still fail to repeat. The failures may be manifested by total inability to repeat, paraphasic repetitions or in many instances the production of an association to the correct word.

A remarkable feature of many of these cases is the frequently preserved ability of the patient to repeat polysyllabic numbers, e.g. 'seventy-eight', while he fails to repeat even shorter words or repeats them paraphasically. The contrast is brought out sharply by such phrases as, 'fifty-five per cent' where our patient said, "fifty-five progum" and 'eleven plus eight' where the patient would say, "eleven, eight... nineteen" but failed to produce the word 'plus' on repeated trials. 'Three-quarters' was repeated as 'three-four' for several weeks. Although the patient could say the series, "Penny, nickel, dime, quarter," he could not use the word quarter in repeating 'three-quarters'. Another patient said 'fifty' for 'one-half' and 'seventy-five' for 'three-quarters'. Even when these patients fail to repaet numbers correctly, their errors are different from those with words. Thus, the patient tends to substitute other numbers but rarely to produce grossly paraphasic responses and even more rarely not to repeat at all. Thus, the patient may say 'six, eight' for 'sixty-eight' or 'nineteen seven' for 'ninety-seven'. This advantage of numbers is not confined to the spoken modality since our first patient read printed words paraphasically but read numbers

aloud correctly whether printed as numerals or as words. Thus, he read 'train' for 'travel', but correctly read 'twenty-eight' just as easily as '28'. It was very striking in this patient that whenever he was presented with numbers, he manifested an immediate and obvious relaxation of effort.

The mechanism of conduction aphasia was discussed by Wernicke (1874). He assumed a lesion in the connexions between what later came to be called Wernicke's area in the first temporal gyrus and Broca's area in the third frontal gyrus. He assumed that these connexions ran in the insula and called this 'aphasia of the insular region'. Some thirty years later Wernicke (1908) discussed this problem again. He stated here that the autopsy findings had not confirmed his view that conduction aphasia was the result of lesions of the insula. He points out later on in this paper von Monakow's emphasis on the importance of the fasciculus arcuatus. This tract runs from the posterior superior temporal region, arches around the posterior end of the Sylvian fissure and then runs forward in the lower parietal lobe, eventually to reach the frontal lobe, and in particular Broca's area.

Kurt Goldstein (1927, 1948) discussed this condition in great detail. He called it not 'conduction aphasia' but 'central aphasia' and defined it as "a speech disturbance, which, in the presence of relatively intact comprehension of speech, is characterized by a disturbance in repetition, paraphasic manifestations in spontaneous speech, reading and writing, disturbance in spelling...." He goes on to say that it appears with lesions between the sensory and motor speech zones.[24]

Goldstein mentions several theories of this condition. Liepmann and Pappenheim (cited by Goldstein, 1927) thought that this syndrome was only a milder variant of ordinary sensory aphasia. Goldstein points out, however, that most of Wernicke's area was intact in their case. Goldstein himself supported Wernicke's original view that it was a lesion of the insula which caused this syndrome and that it was actually the insular cortex and short association fibres which were involved. He did not, however, stress the disconnexion between Wernicke's and Broca's areas that Wernicke had postulated.

Pötzl and Stengel (1936) pointed out in a highly detailed study the interesting combination of conduction aphasia and pain asymboly (I have discussed the latter condition earlier in this paper). They point out the sparing of the insula in several cases of this condition such as the classic case of Liepmann and Pappenheim. In addition, in their own case the

lesion did not directly destroy the arcuate fasciculus. They point out that the lesion common to their case and other earlier cases was the involvement of the left Heschl's gyrus and the planum temporale. Their own case had in addition a lesion of posterior insula and supramarginal gyrus; these as well as the other lesions were purely cortical. They theorized that their lesions probably destroyed the cells of origin of the pathway connecting the sensory and motor speech regions which they assumed to run both via the arcuate fasciculus and the insula. In order to test this theory a more detailed knowledge of the precise cells of origin of the connexions from upper temporal lobe to Broca's area and other opercular regions is needed. One interesting feature of this case was the cortical destruction of the supramarginal gyrus. Since there is considerable evidence that the arcuate fasciculus consists in great part of short fibres, it is not at all unlikely that this lesion might in effect destroy part of the pathway. They attributed, in keeping with the discussion presented earlier, the pain asymboly to the supramarginal gyrus lesion.

Konorski *et al.* (1961) have, as I have noted, revived interest in the problem of conduction aphasia and championed the idea that conduction aphasia results from a lesion of the arcuate fasciculus. I would think that in the light of the preceding discussion their view is probably correct.

It has been suggested that the development of language in man depends on his possession of an arcuate fasciculus, while a monkey does not have this pathway. This seems at first a reasonable conclusion from the findings of Bailey *et al.* (1943a) who show no arcuate fasciculus in their diagram of the long association fibres in the chimpanzee. There are, however, several reasons for rejecting this conclusion: (1) They were studying only *long* association fibres. Since it is possible, as we have noted earlier that much of the arcuate fasciculus consists of short fibres (Crosby *et al.*, 1962), these would not have been demonstrated in the study of Bailey *et al.* (2) There are, however, also long arcuate fibres revealed in later studies by some of Bailey's pupils (Sugar, French and Chusid, 1948) which demonstrated unquestioned evidence of fibres from the temporal operculum to the parietal and frontal opercula. (Their diagram does not show these fibres arching around the back end of the Sylvian fissure. Since they were using strychnine neuronography, there would have been no way for them to know the course of the fibres involved but only the origins and terminations.) These had been missed in the earlier study because Bailey *et al.* had not explored

the depths of the sulci. The study of Sugar *et al.* certainly proves the origin of such fibres in the supratemporal plane. Whether these definitely arise from primary auditory cortex or from association cortex is not completely clear because of the great crowding of these structures in the small primate brain. (3) Bailey and Bonin (1951) themselves state, "Physiological neuronography presents evidence for both uncinate and arcuate bundles.... Whether the firing of the inferior frontal gyrus and the parietal operculum by strychninization of the first temporal convolution, that of the inferior frontal gyrus from the second and of the middle frontal gyrus from the inferior temporal gyrus (Petr *et al.*, 1949) is due to conductions in the arcuate or uncinate fascicle cannot be decided at present; that the last, if not the latter two, observations should be ascribed to the arcuate fascicle seems more plausible. The firing of the middle temporal gyrus from the inferior parietal lobule as well as the firings of the inferior parietal lobule from the inferior frontal gyrus can be taken as further evidence for 'arcuate' fibres."

There is one further theory of the mechanism of conduction aphasia, one first mentioned by Liepmann and Pappenheim but defended most recently by Kleist (1962). This author argued that conduction aphasia occurs in patients in whom there has been actual destruction of the left temporal speech area itself. This lesion would in most instances lead to profound comprehension defect but does not do so in certain people because their right temporal region can take over this function. He assumes that these patients, however, still must rely on the left Broca's area for speech. The pathway from the right temporal speech area to the left Broca's area is, however, interrupted in these patients by the destruction of the left temporal lobe and therefore repetition is poor. Kleist presents 4 cases in support of this thesis. The first case, Spratt, is particularly striking since the destruction of the left superior temporal region was indeed so extensive as to be expected to give rise to profound incomprehension. This case would appear to be strong evidence for Kleist's contention. It is further of interest that this patient was ambidextrous, using his left hand for many complex functions. By contrast the lesion of his fourth case, Treusch, involved the left temporal speech area very little and appeared to involve deep white matter, particularly in the inferior parietal lobule. This case would appear to be much better evidence for the theory of involvement of arcuate fasciculus than for Kleist's own theory. In fact, Kleist's own

diagram implies that even if the mechanism he suggests is correct in some cases, involvement of arcuate fasciculus should still produce the same syndrome. In summary, it appears that Kleist has good evidence for his mechanism in at least some cases of conduction aphasia. I would think that if there are two forms of the disorder, they might well be distinguishable clinically. We are attempting in our own cases of conduction aphasia to ascertain whether the right hemisphere really plays a role in any of them, as asserted by Kleist.

A brief note is appropriate on the already mentioned observation that number repetition is better than word repetition in many of these cases. It is conceivable that this is the result of sparing of a different anatomical pathway for numbers. It would seem unlikely, however, that numbers are normally spoken over a different pathway from words. I believe that a more likely explanation comes from a consideration of how any repetition is carried out in these patients. Thus, although the patient fails to repeat the correct word he is likely to give an association of this word. In fact, in some cases when the correct word itself is repeated, it is likely that this has been accomplished only by way of associations. One patient on being given his own last name first replied with the given names of his brothers and himself and then finally gave the family name. Had the intervening names not been spoken aloud the fact that the eventual correct repetition was by way of associations and not 'direct' could easily have been overlooked. This type of associative repetition probably does depend on finding a 'detour' around the damaged normal direct pathway from Wernicke's to Broca's areas. It is natural that this should lead to many errors in repetition of *words* which in general arouse a large reservoir of associations. The associations to numbers are likely to be more limited, and in fact are not at all unlikely to be confined to the number itself. The errors in number repetition support this theory; thus, 'six, eight' for 'sixty-eight' and even more dramatically 'three-four' for 'three-quarters', 'fifty' for 'one-half' and 'seventy-five' for 'three-quarters'. The marked difficulty in nonsense syllable repetition stressed by Konorski would result from the paucity of associations to such material. In brief, I think that these patients probably do not repeat either numbers or words via the normally used pathways but that in response to a heard number or word, the patient gives an associative response; the associative response is more likely to resemble the original if this is a number but not a word. One might say

that there is 'pseudo-repetition' of numbers. It is interesting that, as pointed out by Dr. Susan Ervin (personal communication), 'parrot-like repetition' (i.e. precise repetition of what has been said) is a late development in children acquiring language. Contrary to ordinary views such precise repetition may well be an advanced activity dependent on specialized use of the pathways between the posterior and anterior speech regions.

2. *The Case of Bonhoeffer*

Bonhoeffer (1914) described a remarkable case which, although it presents certain difficulties in interpretation, I believe is worth presenting here. Although it is apparently unique, I suspect that other such cases have been overlooked by other observers[25]. Bonhoeffer's patient sustained a transient right hemiplegia, the arm subsequently improving faster than the leg. There was, however, permanent reduction of speech to one or two words; verbal comprehension was much better preserved but reading and writing were both very poor. The patient showed difficulties in carrying out verbal commands, in imitation and in object handling, all these disturbances being much more marked on the left than on the right. At postmortem there was an infarct involving the posterior parts of the left superior and middle frontal gyri and the anterior four-fifths of the left side of the corpus callosum. There was an infarct of the anterior limb of the left internal capsule; finally a small lesion was present under the left parieto-occipital region. The cause of death was a fresh haemorrhage in the lenticular nucleus on the right but the right hemisphere was otherwise intact. For obvious reasons Bonhoeffer attributed the severe apraxia of the left arm to the callosal lesion. To explain the aphasic disturbance of verbal expression, he pointed out that the infarct of the anterior part of the internal capsule had cut the descending pathways from the speech area. The transient hemiplegia was, of course, probably due to transitory neighbourhood effects of this lesion. This capsular lesion alone would, of course, produce no aphasia; nor would the frontal lesion which clearly spared Broca's area. Bonhoeffer pointed out that normally no aphasia is seen with a capsular lesion. In the presence of such a lesion an outflow path still exists from Broca's area running across the callosum to the corresponding cortical area on the right, and from there eventually reaching the right internal capsule. The callosal infarct had, however, cut off this alternative pathway and Broca's area was isolated.

My own inclination would be to agree on the whole with this interpretation of Bonhoeffer's. There remain to be explained the mild rightsided apraxia as well as the alexia and agraphia. Bonhoeffer himself was uncertain as to their cause, but I would regard them as having been caused by the lesion of left motor association cortex as well as by the lesion under the left angular gyrus. It is important to note that the latter was a small lesion which Bonhoeffer felt would not be very significant clinically. Bonhoeffer, however, then goes on to express his agreement with von Monakow's view that combinations of lesions may produce clinical pictures that none of the lesions could cause in isolation. (I have, of course, given several examples of such combinations.) The left angular gyrus lesion may have been much more disabling in this patient because of the co-existing extensive callosal lesion, which must have cut off most of the callosal connexions of the parietal association areas.

3. *Echolalia*

Kurt Goldstein (1917, 1948) has shown great interest in this symptom; Stengel (1947) has written an excellent paper on the functional aspects of echolalia. A characteristic clinical picture is that of *marked* preservation of repetition, indeed automatic repetition, in the face of gross difficulty in comprehension. I would stress that what is being spoken of here is a retention of repetition which is in sharp contrast to the disturbance in comprehension; other explanations probably are forthcoming for the common situations in which repetition is only moderately better than comprehension. To explain the sparing of repetition in such cases on the basis that it is 'simpler' runs counter to the fact that repetition can be especially impaired, as in conduction aphasia.

Goldstein stressed that for this syndrome to be present it was necessary that Wernicke's area, Broca's area and the connexions between them be intact. This intactness of the speech area and of its afferent auditory connexions guarantees repetition. Coupled with thus intact speech area there must be a lesion which isolates the speech area from much of the rest of the cerebral hemispheres, i.e. a large parietal lesion. It is the *isolation* of the speech area which tends to produce this picture. There is no comprehension because language arouses no associations; there is gross disturbance of spontaneous speech since the speech area receives no information from elsewhere in the brain.

Segarra and Quadfasel (1961) have recorded the post-mortem findings of a most interesting patient who had been studied extensively by Quadfasel. This patient, a woman of 32 years of age, suffered severe illuminating gas poisoning, following which she survived for almost ten years. During this period she never showed any evidence of comprehension of language, nor ever spoke spontaneously a propositional phrase, but uttered only expletives. She could do nothing for herself. By contrast, she echoed what was spoken to her and even more strikingly she echoed songs. In fact, she was *even able to learn new songs not in existence before the onset of her illness*. Following her death her brain was cut in whole-brain sections and stained for cells and myelin. Preliminary survey of these sections has shown intactness of the speech area (Wernicke's and Broca's areas) and of the arcuate fasciculus and insula. In addition the hippocampal region is intact as well as connexions to it within the temporal lobe. The corresponding structures on the right side are symmetrically preserved. By contrast there is extensive infarction extending in mantle form around these intact regions. This patient had essentially an intact speech area which was able to carry on repetition. It would even manifest verbal learning because of the preservation of the connexions to the hippocampal region. Comprehension and propositional speech were, however, lacking for the reasons which I have already presented.

Cases of isolation of the speech area such as these are most valuable in elucidating the functions of this region. It is most remarkable that this patient could learn verbal material. This task is intensely difficult for patients with partial lesions *within* the speech area who may show syndromes far less devastating than that revealed by this patient. It also suggests that language is not 'comprehended' by Wernicke's area but rather that this region serves to arouse associations elsewhere probably by way of the inferior parietal region. It illustrates also that speech is not created in Wernicke's area; rather it serves to transform what has come from the remainder of the brain into language.

VII. POSSIBLE OBJECTIONS AND PITFALLS

1. *The Results of Akelaitis and His Co-workers*

I would like to deal briefly here with the objections that might be raised against this theory of the importance of lesions of association cortex and

association fibres in producing disturbances of the higher functions of the nervous system. These objections arise chiefly from the results of the generally negative investigations made by Akelaitis and his co-workers (Akelaitis, 1941a, 1941b, 1941c, 1942a, 1942b, 1943, 1944, 1945; Akelaitis *et al.*, 1942) in cases with surgical section of the corpus callosum for the treatment of epilepsy. I have discussed this problem briefly elsewhere (Geschwind, 1962). I believe that the patients were well examined; inadequate examination is therefore probably not the explanation for the discrepancies between Akelaitis's results and many of those which I have cited.

The majority of Akelaitis's patients had had cerebral lesions dating from early childhood. Such early lesions might well explain the absence of cerebral dominance for language. Thus, many of the functions which are normally unilateral could probably be carried on by both hemispheres. A similar explanation probably applies to the lack of such syndromes in cases of agenesis of the corpus callosum. The early lesion is more likely to lead to functional re-organization than lesions in adult life (as examples of this one may cite the work of Kennard, 1942 and of Scharlock *et al.*, 1963), i.e. it is more likely to lead to use of other pathways.

A second factor is the presence of epilepsy in nearly every case in Akelaitis's series. It is certainly highly possible that seizures may lead to the 'learning' of new pathways (I have commented earlier in the paper on Morrell's work on the parallels between epilepsy and other learning). Similarly, Penfield and Boldrey (1939) wrote, "An habitual seizure, by virtue of its frequent repetition, may eventually establish a complicated neurone pattern.... (The spread of a seizure) is not diffuse but... along a definite neurone system which may be preformed or acquired. By 'preformed' is meant a system of neuronal connexions ordinarily recognized as physiologically functional in normal brains. By 'acquired' is meant a pattern of neuronal connexions established by the conditioning influence of previous experience.... In complicated seizures the advance of discharge along this neurone pattern may be so slow and episodic as to suggest that isolated ganglionic collections are fired in an advancing series...." The general failure of callosal section as a therapeutic agent in Akelaitis's cases is compatible with the seizures having long since 'learned' some complex or unusual pathways or having 'learned' such new pathways after the transection. Erickson (1940) pointed out that section of the corpus cal-

losum in monkeys prevented electrographic spread of the seizure to the opposite hemisphere and altered the pattern of the seizure. Whether electrographic spread to the opposite hemisphere might have continued to take place despite transection of the corpus callosum if the experiments had been done on chronically epileptic monkeys rather than in acute experiments is not known. Such an experiment would, however, be of great interest in determining whether seizures 'open' less commonly used pathways to the other hemisphere. The second part of Erickson's experiment seems to confirm strongly that with a lesion confined within the bounds of a hemisphere seizures rapidly 'find' new paths to circumvent the obstruction. Thus, in one monkey a seizure initiated in the right cortical arm area spread in the intact brain to the right leg area, left leg area, and left arm area. After a wide section at right angles to the Rolandic cortex superior to the right arm area, the seizure travelled from the right arm area to the left arm area then to the left leg area and finally to the right leg area. Section of the callosum abolished this new mode of spread.[26]

As an example of the joint effects of seizures and early lesions, we may compare the cases reported by Akelaitis in whom there had been section of the splenium, and those reported by other authors. There were 6 such cases in Akelaitis's series, all of them severely epileptic, 5 with lesions from early childhood. None developed alexia in the left visual field following section of the splenium. By contrast the case of Trescher and Ford (1937) and the 2 cases of Maspes (1948) all had colloid cysts of the third ventricle and none had had seizures. All three developed alexia in the left visual field as a consequence of having had the splenium sectioned.

The notion that repeated seizures favour the opening of less used pathways leads to several interesting implications. One should be able in experimental animals to overcome the effects of disconnecting lesions by repeated seizures. Thus, an animal with the callosum sectioned who has had repeated seizures for a long period before callosal section might show interhemispheric transfer after surgery while a control without seizures might not. It goes without saying that the long-term effects of section of the callosum should be less disturbing if carried out in infant animals than in adults.

It is remotely possible that the effects of repeated seizures in favouring the opening of new pathways, if verified, might be useful therapeutically. Repeated induced seizures might favour recovery from disturbances fol-

lowing lesions of association cortex or fibres. Obviously, such a therapeutic investigation could be justified only after experimentation had confirmed the effectiveness of such procedures in animals.[27]

There are two other but less likely explanations for Akelaitis's results. One is that alternative pathways to the opposite hemisphere via subcortical routes are readily available in most people and that these syndromes therefore appear only exceptionally. The other is that the syndromes had cleared by the time the patients were tested. Although the patient of Geschwind and Kaplan (1962) did show definite improvement in his callosal disconnexion symptomatology over several months, it was still evident after this period. Furthermore, acquired lesions of many other association pathways may show no improvement with time. Restitution after white matter lesions might conceivably be less damaging permanently than lesions of the corresponding cells of origin; as long as the cells are intact, there is a possibility that collateral pathways might be brought into play. We know, however, almost nothing of the pathways of secondary importance.

This leads us to the greatest advantage but at the same time the greatest danger of thinking in terms of disconnexions. Theories of this type are rich in readily testable theoretical implications. They can readily degenerate into naïvely anatomical systems. This is, however, not inherent in the structure of this approach and there is no conflict between this kind of reasoning and sophisticated psychological analysis. In fact, this approach frequently demands a reinvestigation of standard psychological categories and opens new ways of looking at psychological phenomena. Perhaps the greatest danger is that of 'working backwards' and of inventing pathways to correspond to every difference in behaviour. I have tried to show here that while some differences in behaviour are probably based on anatomically different pathways (e.g. the discussion on isolated limb movements versus whole body movements) others are dependent on non-anatomical factors (e.g. the discussion on the relative preservation of number repetition in conduction aphasia). I can only agree with the hope expressed by Adolf Meyer (1905) for "convincing observations of patients... with such anatomical examination as will put an end to the regrettable tendency of so many clinicians to consider the white matter of the hemispheres the cornucopia of all the desirable conduction paths."

VIII. PHILOSOPHICAL IMPLICATIONS

It is not my intention to embark on an extensive philosophical discussion in this section. It seems reasonable, however, to sketch rapidly the implications of many of the results discussed here for the philosophical foundations of study of the higher functions of the nervous system.

1. *The Whole Man*

For the past forty years there have been schools of thought which have stressed the importance of thinking of the patient as a whole, of seeing his responses as those of an integrated unitary structure, even in the face of damage. The ramifications of this thinking in neurology, psychiatry, psychology and other fields must be well known to most readers. It should be clear from much of our discussion that this principle, while it may be useful in some cases as a stimulus, may be actively misleading when it is regarded as a philosophical law. When Edith Kaplan and I were studying our patient, we constantly found that many confusions about the patient in our own minds as well as those of others resulted from failure to do the exact opposite of what the rule to look at the patient as a whole demanded, i.e. from our failure to regard the patient as made of connected parts rather than as an indissoluble whole. We were constantly dealing with questions such as "If he can speak normally and he knows what he's holding in his left hand why can't he tell you?" We had to point out that we couldn't say that "the patient knew what was in his left hand" and that "the patient could speak normally," since that part of the patient which could speak normally was not the same part of the patient which 'knew' (non-verbally) what was in the left hand. This is at first blush an odd way to speak – it is hard not to say 'the patient' and yet it is clear that this terminology is misleading.

We have little difficulty with the concept of disconnexion at lower levels. If the spinal cord is transected we are usually capable of treating it as separate from the higher centres. We may say of such a person, "The patient urinated" since we know that in this case this means "The patient urinated involuntarily." Although 'the patient urinated' is at first glance ambiguous, in practice we understand and do not find it necessary to use clumsy locutions such as "The patient's spinal cord urinated." We get into difficulties, however, with disconnexions at higher levels since we do not

expect highly organized activities dependent on learning to be carried on in disconnected parts of the brain. We must become accustomed to thinking in this way in order to understand some of the more complex disturbances consequent on lesions of the brain. I am not advancing 'the atomistic approach' as a basic philosophical postulate to replace 'the holistic approach', but am rather suggesting that failure to consider the applicability of *either* type of analysis will in one situation or another lead to errors. It should be pointed out that the usefulness of sometimes considering animals or humans not as a unit but as a union of loosely joined wholes need not apply only to disease states although probably it will find its greatest use in that situation. Probably even in the normal person parts of the brain are so weakly connected as to make their interaction difficult. I have suggested that some connexions are normally present in adult man which are absent or of less extent in lower animals; these connexions may take years to develop in some children. Perhaps an adult man is more unitary than a chimpanzee, but perhaps total unity is never obtainable because of the necessary separation of some structures.

2. *The Unity of Consciousness*

A corollary to the above discussion is that it forces us to be somewhat more precise than we have been about 'the unity of consciousness'. It would no doubt be startling to suggest that the patient of Geschwind and Kaplan had separate consciousness in each hemisphere; it would on the other hand be a little difficult to understand just what would be meant by saying that his consciousness was unitary. If the ability to give a verbal account is a prerequisite of consciousness then only the left hemisphere was conscious; if the ability to respond in a highly organized manner and to use the results of past experience constitutes consciousness then he had multiple consciousness. Perhaps there are better criteria of consciousness than these; this case and similar ones only re-emphasize the necessity of re-evaluating the idea of the unity of consciousness if it is to be at all useful.

I believe that Kurt Goldstein was perhaps the first to stress the non-unity of the personality in patients with callosal section and its possible psychiatric effects. Thus he wrote (Goldstein, 1927), "The separation of so large a part of the brain and the resulting impossibility of evaluating stimuli perceived with the right hemisphere... surely cannot be without

effect on the total personality.... I have pointed out the presence in my patient of a feeling of strangeness in relation to movements of the left hand, which she described with such curious expressions (she would say that someone was moving her hand and that she wasn't doing it herself) that she was regarded at first as a paranoiac. It appears to me not to be excluded that on this basis and under certain conditions there may develop paranoid states, perhaps also the experience of doubled personality and above all the experience of being influenced from without...."

3. *The Value of Introspection*

Still another corollary to the observation that parts of the brain may be disconnected is the conclusion that introspection may be an extremely ineffective way of obtaining information about many of the patient's experiences. I have already made this point in the presentation of some of the syndromes of disconnexion, e.g. in respect of the colour-naming disturbance of pure alexia without agraphia. If a part of the brain is fully disconnected from the speech area it will not be possible for the speech area to give an account of what goes on in that part of the brain. The patient with a colour-naming disturbance can give only a poor account of his colour-experience or indeed none at all since his speech area has little or no access to information about the colour-experiences of the visual cortex. Other examples were cited illustrating the same principle. I have also discussed in some detail the tendency for the patient to show confabulatory response to demands for introspective observations. It is certainly well known that even in normals introspection may be misleading or incorrect.

Let me point out carefully that what is presented here is not the gross behaviouristic assertion that introspective information is in all situations useless nor that one should never listen to what the patient is saying. The conclusion being presented is the less extensive one that there are certain situations, particularly in the presence of lesions of the brain, in which the patient for simple anatomical reasons is incapable of recounting verbally the experiences of parts of his nervous system which are functioning at a complex discriminative level.

4. *Language and Thought*

An old problem is the one of the extent to which language controls one's

'perception' of the world. Whorf (1956) suggested an extreme form of the view that language influences what is perceived. The view at the opposite extreme is the naïve 'natural' one that language is an infinitely flexible tool for describing without prejudice the impact of the world on one's nervous system. The behaviour of our patient with colour-naming defect is of interest here. Despite his inability to name colours correctly, he had no difficulty in sorting, and did so by colour. This, of course, does not necessarily reflect the behaviour of a 'naïve' nervous system since the visual region may have been trained by earlier verbal experience. It is clear at last that words need not be available for the sorting process to take place.[28] On the other hand, as I noted in my discussion of secondary sorting errors, the use of the incorrect word may lead to errors in sorting under the conditions of certain types of instruction. This type of 'secondary' error might be more marked in tests where the subject is required to remember colours after an interval since it seems reasonable that such memory is frequently mediated verbally (Brown and Lenneberg, 1954). The study of patients with such specialized disturbances may aid in further exploring the relationship of language to cognition.

NOTES

[1] It will perhaps strike the reader as curious that Goldstein should have embraced so classical an approach. I have discussed more fully elsewhere Goldstein's position in the history of aphasia (Geschwind, 1964a) but can only comment here that he was in fact much more of a localizationist than is generally appreciated. His theoretical writings with their criticisms of classical ideas contain so many qualifications that they are often compatible with even the most extreme localizationist views. The reader who goes carefully through the 'Special Part' of Goldstein's 1927 monograph will repeatedly find in it an active defence of many classical ideas; indeed, many of Goldstein's disagreements with other authors are primarily on details of localization. His later book on language (Goldstein, 1948) continues to show his acceptance of many classical ideas, especially in his discussion of particular syndromes.

[2] In the monkey there appears to be a bundle which is homologous to this structure in the rabbit and which perforates the splenium of the corpus callosum. There is, however, some question as to whether the bundle even in the rabbit does indeed rise in the visual cortex; for the monkey the site of origin of the corresponding tract is unknown, and it is certainly possible that it arises from retrosplenial cortex or from areas 18 or 19 on the medial surface. It is conceivable that this bundle may run directly from the visual cortex to the hippocampus in the rabbit while in higher forms these connexions must be made by way of association cortex. The analysis by Pribram and MacLean (1953) of the connexions of the mediobasal cortex of the monkey presents ample evidence for *indirect* pathways which could lead along the medial surface of the hemisphere from visual cortex to visual association cortex, hippocampal gyrus and hippocampus.

[3] Petr *et al.* use the term 'fusiform gyrus' for areas TF and even TH in the macaque. Bonin and Bailey (1947) note the great similarities of TF and TH. Papez (1929) uses the term 'fusiform-hippocampal gyrus' and 'pyriform area' for these two regions. It is likely that the fusiform gyrus in the human sense is not present in the monkey and that these two areas are probably most reasonably considered as hippocampal gyrus (now called parahippocampal gyrus by some authors). Whitlock and Nauta (1956) in reporting the results of Petr *et al.* substitute the term 'hippocampal gyrus' for 'fusiform gyrus' and we will follow their usage.

[4] I use this term advisedly rather than 'loss of visual discriminations.' The subsequent theoretical discussion will make the reason for this choice of words clear.

[5] These elementary results have an important bearing on the question of cross-modal associations. Recent studies, e.g. those of Ettlinger and different co-workers have clearly demonstrated difficulties of tactile-visual or visual-auditory transfers in monkeys (Burton and Ettlinger, 1960; Ettlinger, 1961). The conclusion should, however, not be drawn that monkeys form no cross-modal associations. It is abundantly clear, in fact, that the majority of classical learning experiments with most organisms do in fact demonstrate formation of cross-modal associations, as long as the modality to which the association is made is a 'limbic' modality, i.e. a reinforcer, such as food, water, etc. I will return to the question of the difficulty of transfer between 'non-limbic' modalities at a later point.

[6] The authors state that they ablated 'areas 18 and 19', but it is possible from their diagram that part of the association cortex posterior to the lunate sulcus was preserved. Bonin and Bailey (1947) place OB behind the lunate sulcus and Crosby *et al.* (1962) show area 18 as behind the lunate sulcus. It is not unlikely that some of area 18 was spared in the Ades and Raab experiments.

[7] Since the writing of this section it has been called to the author's attention that other experimenters have had results different from those of Downer. It will be important to ascertain the reasons for these discrepancies.

[8] Meyer and Yates (1955) showed that patients with left temporal lobe lesions are likely to have verbal recall difficulties. Milner (1962) has confirmed their findings, showing that left anterior temporal lobectomy has a more profound effect on verbal memory than similar right temporal lobectomies. It is quite likely that the reason for this is that left temporal lobectomy cuts off connexions between the posterior speech area and the limbic system and thus leads to verbal learning deficit.

[9] This mechanism for incomprehension of spelled words appears to Dr. Howes and myself to be more simply and more clearly based physiologically than the classical explanation, which simply invokes a new disturbance, 'word-sound deafness', to account for incomprehension of spelled words. By any standard the term 'word-sound deafness' is a poor one. 'Letter-name deafness' would have been closer to being a correct description. 'Inability to understand words spelled orally' is the best descriptive term.

[10] I am indebted to Sir Charles Symonds for having called Vialet's monograph to my attention. It was in fact his paper (Symonds, 1953) which alerted me to this interesting syndrome. I am also grateful to him for having read and criticized an earlier paper of mine on this topic.

[11] It may be objected that the alexia in a half-field from a right parietal lesion is the result of 'neglect' of that field. While I do not wish to discuss this problem extensively here, I would like to point out that what I am attempting to show is that one mechanism of 'neglect' of a normal left visual field is disconnexion of the normal right occipital cortex from the speech area.

[12] In an illiterate society a lack of visual-auditory associations would not seriously inconvenience anyone except in unusual situations; literacy makes this ability highly important. Other cross-modal association deficits may exist but might never be detected because they cause so little disturbance. It is conceivable that direct visual-tactile associations may be as badly developed in many humans as they appear to be in monkeys (Ettlinger, 1960) but only specific testing will bring this out. It is important, of course, to study children as early as possible in the course of development.

[13] These authors also found another group of parietal areas which responded to click with only slightly longer latency than the primary auditory region. They presented evidence that the response in these areas depended on collaterals from the medial geniculate body. These areas would not in my terms be 'association' areas. I will not discuss their possible function here.

[14] It should be added that the second temporal gyrus of man appears to be a phylogenetically very late region of whose connexions we know very little. It may be a region of great importance and it is conceivable that the view of Wernicke's area presented above is too narrow. I would, however, disagree with those authors who include in Wernicke's area all the posterior regions involved in speech in both the temporal and parietal lobes.

[15] Dr Edwin Weinstein and his co-workers have been instrumental in stressing in recent years in a series of careful papers the importance of confabulatory responses. The stimulation of his work was a major factor in attracting my attention to this problem.

[16] Pick (1931) ponted out that sorting errors may result from difficulties in verbal mediation in patients with colour-naming troubles.

[17] A brief note is appropriate here on Nielsen's interesting and important review. In his introduction he points out that many authors have stressed the bilaterality of lesions in most cases of this disorder. He then goes on in his study to confine himself only to the unilateral cases on the grounds that "if in any case a unilateral lesion produced visual agnosia, there can be no point in citing cases of bilateral lesion of the occipital lobes." This argument is of course based on the reasoning that a bilateral case must only be a unilateral case with an extra unnecessary lesion. The fact that probably the *majority* of cases of visual 'agnosia' have had bilateral lesions militates against this thesis. Furthermore it should always be kept in mind that many different *combinations* of lesions may lead to the same effect when the physiological mechanisms are kept in mind. He also neglects the possibility of individual differences. The paper of Nielsen is therefore a contribution to the anatomy of the unilateral group of cases but not necessarily to that of the bilateral cases. Many of Nielsen's cases show the discrepancies between naming and other functions that I have cited above although his interpretation of much of the material is different from mine because he accepts the idea of defects of 'recognition'. He frequently uses as one of his criteria the failure of the patient to *behave correctly* in relation to the 'unrecognized' stimuli; at the end of this section on the agnosias I will discuss this problem briefly.

[18] Although I have leaned heavily on the work of Weinstein and his co-workers in these preceding statements, I would by no means wish to suggest that Dr. Weinstein would have used the phraseology I use here.

[19] It should be noted here that Denny-Brown (1962) also believes the left hemisphere to be generally dominant and rejects right parietal dominance but on quite a different basis from that presented here. Since the initial writing of this paper my attention has been called to the results of Kimura (cited by Shankweiler, 1964 and confirmed by him) who found that in normals melodies were better recognized when presented to the *left*

ear (in contrast to the greater efficiency of the handling of digits by the *right* ear). These differences in melody recognition, although significant, were small. This suggests that although the right hemisphere may be more *efficient* in certain perceptual tasks, it is not *dominant* in the sense that the left hemisphere is dominant for speech. This view is further supported by Shankweiler's observations that even the presence of a right epileptogenic lesion shifts the balance in favor of the right ear in melody recognition. In any case even the confirmation of some greater efficiency of the right hemisphere in certain tasks would not invalidate the mechanisms I have suggested for some of the dramatic right-hemisphere syndromes.

[20] Weinstein, Cole and Mitchell (1963) argue that denial is less common in left-sided lesions because speech is less available; while this view, in my opinion, explains many of the phenomena, it does not deal fully with nonverbal aspects. To round out the theory it must be assumed, as I have already commented, that left dominance is for more than speech.

[21] The italics are mine (N.G.).

[22] Let me stress that I am confining the term apraxia to the sense in which Liepmann used it. I am not including the cases separately tabulated by Ajuriaguerra *et al.* as 'constructional apraxia' and 'dressing apraxia'.

[23] Some authors have, however, denied the existence of this tract. Thus, Whitlock and Nauta (1956) mention that after placing lesions in the temporal lobe of monkeys, they found no degeneration running to the pons. These authors themselves admit, however, that their material did not include lesions in the more caudal temporal regions as is apparent from inspection of their diagrams. This explanation may apply to other instances of failure to demonstrate a temporo-pontine tract. Bucy and Klüver (1955) also strongly deny the existence of this tract but also had no case of a posterior temporal lesion. By contrast, in support of Brodal's view, Krieg (1963) points out that in the macaque the only downward connexion of area 22 goes to the lateral cells of the pons. It is clear that this problem deserves careful reinvestigation.

[24] It should be noted that Goldstein (1927) explicitly equates his central aphasia to conduction aphasia. Thus he writes, "The symptom-picture which is usually designated as conduction aphasia.... *I called it central aphasia*...." The descriptions by Brain (1961) and Russell and Espir (1961) appear to me not to make this distinction and to intermix elements of other aphasias. Neither of these authors stresses the component of disturbed repetition with relatively preserved comprehension which is the essence of this disorder. In his later writings Goldstein (1948) continues to use this earlier definition; he writes, "Understanding is usually preserved best, repetition and spontaneous speech are always severely damaged...."

[25] In fact, after having read Bonhoeffer's observation, I have come to suspect that a case I saw several years ago probably was an example of the same disturbance but that I completely missed its significance at the time. Chance favours, as is well known, the prepared mind; Bonhoeffer's was obviously very well prepared for this patient.

[26] The term 'arm area' does not necessarily mean area 4; the electrical disturbance was probably, in my opinion, going via area 6. This discussion is not meant to suggest that seizures spread only between cortical regions. Certainly some of Erickson's observations in the above paper confirmed that bilateral involvement of the body can occur with the callosum sectioned – but this is different in type from the kind of bilateral spread seen when the callosum is present. Obviously bilateral spread with the callosum sectioned depends on involvement of subcortical structures. Gastaut and Fischer-Williams (1959) have discussed such subcortical spread extensively in their review. One of the

major reasons for the failure of surgery in Akelaitis's patients may have been such spread to subcortical motor systems rather than spread to the opposite hemisphere via 'new' subcortical pathways. We do not have enough information to decide which is the explanation.

[27] To forestall any questions in advance I am in no way suggesting that the effects of electric shock therapy are related to such a mechanism!

[28] Some informal experiments on children suggest that perceptual differentiations precede naming. My 4-year-old son misnamed colours but could sort correctly and could trace out correct numbers on the Ishihara test; although I could name the colours of dots, I did much poorly than he did on these tests. His difficulty was on of colour-naming, mine was partial colour-blindness.

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CHAPTER IX

COLOR-NAMING DEFECTS IN ASSOCIATION WITH ALEXIA

PREFACE

The case presented in this paper was called to my attention by Dr. Michael Fusillo, and was another case of alexia without agraphia with some special features. I might point out that this syndrome has turned out to be remarkably common, despite the fact that only a few years ago its very existence was questioned.

The analysis of the defect in the naming of colors in this case would not have been possible without the notion of disconnection. I realize now that we really did not use the most appropriate name for the disorder. To say that the patient could not name colors is not quite accurate, since he could find the names in response to such questions as "What is the color of a banana?" A more accurate description would have been 'inability to match a seen color to its spoken name'.

One effect of the disconnection approach has been to force precise statements as to the reasons for the presence or abence of effects after a lesion. Without such a clearly-stated theory the pattern of lost and retained abilities would merely constitute a mystery. This is not to suggest that we understand all the reasons for the pattern of loss and preservation, but that we can at least formulate an approach to their clarification. Thus the type of difficulties with color described in this paper occurs in some but certainly not all cases of pure alexia. My own suspicion is we will have to account for this not only by differences in the pattern of lesions, but also by differences in individual response to the same lesion, which will in turn depend most often on genetic differences, although in some cases early experience may be the determining factor.

COLOR-NAMING DEFECTS IN ASSOCIATION WITH ALEXIA*

1966

ABSTRACT. A case history is presented of a patient exhibiting the syndrome of pure alexia without agraphia who also showed marked difficulties in color identification. The patient failed to name seen colors correctly and could not select a color from a group when given its name; in other words, he showed an isolated difficulty in uttering or comprehending the names of seen colors. By contrast, difficulties in color perception were ruled out by the following tests: the patient correctly matched colors by hue, despite great differences in saturation or brightness, correctly matched colored papers to uncolored pictures of objects, and performed without error on two pseudo-isochromatic tests of color vision. Verbal memory for color names was intact since he correctly named objects corresponding to named colors and named colors corresponding to named objects.

It was pointed out that the best characterization of his syndrome is "an inability to match seen colors to their spoken names."

At postmortem the patient showed the classic lesion of pure alexia without agraphia, ie, infarction of the left calcarine cortex and of the splenium of the corpus callosum. The symptomatology is interpreted on the basis of these anatomical findings as a disconnection of the intact right visual cortex from the speech regions. Alternative interpretations are discussed. In particular, we show that the findings in our case cannot be interpreted as resulting from a minimal aphasia combined with a minimal perceptual disturbance.

Disturbances of color identification in association with the syndrome of pure alexia without agraphia have been repeatedly recognized in the past. Lange (1936) reviewed these extensively. More recently Critchley (1965) has reviewed the literature on such disturbances of color identification. He expressed skepticism as to the existence of the syndrome of aphasia for color names and advanced the view that such cases probably represented the combination of a perceptual deficit with a minimal aphasia.

We have recently had the opportunity to clarify the problem of color-name aphasia by repeated study of a patient with the syndrome of pure alexia without agraphia over several months. On the basis of these studies, we believe that the evidence is now clear that a disturbance of color-naming, or more correctly, of matching the spoken name of the color to the seen color, can be shown to exist in the demonstrated absence of any perceptual disturbance. The patient to be described eventually came to post-

mortem examination and demonstrated, as will be pointed out, the classic pathology of the syndrome of pure alexia without agraphia as first described by Dejerine (1892) and repeatedly confirmed since then. A preliminary description of this case has been published (Geschwind and Fusillo, 1964).

I. REPORT OF A CASE

H. C. (MMH 426-89), a 58-year-old machinist had been known to be diabetic for 17 years. He was transferred to the Massachusetts Memorial Hospital (where he was under the care of Dr. Charles Kane) from another hospital on Sept. 24, 1962, because of right-sided weakness, confusion, and difficulty expressing himself of several days' duration. These difficulties had come on abruptly during a hospitalization for reinvestigation of a long-standing severe sensorimotor polyneuropathy.

The past history was not contributory except for long-standing diabetes mellitus and episodes of postural hypotension in the few months preceding his admission. There was no history of hypertension or previous signs or symptoms of cerebral vascular disease.

Lumbar punctures performed at the other hospital on previous admissions had shown on one occasion a protein of 113 mg/100 ml, and on another 330 mg/100 ml. Pressure, cell count, and serology were normal on both occasions. On these admissions serum proteins were normal and a rectal biopsy for amyloidosis was negative. Except for the evidence of diabetes, the results of other laboratory studies were normal. Before admission to the Massachusetts Memorial Hospital the patient's medications had included protamine zinc insulin suspension (60 units per day), chlorpropamide (Diabinese) (25 mg three times per day), thiamine (100 mg daily), and liver extract and cyanocobalamin (Vitamin B_{12}) injections every other day.

General physical examination on admission to the Massachusetts Memorial Hospital revealed a well-developed, well-nourished white man in no acute distress who, although right-handed, showed lack of spontaneous use of the right arm. The vital signs were as follows: blood pressure, 128/78 mm Hg, pulse, 120 beats per minute and regular; respirations 24 breaths per minute and regular; and temperature, 98.6° F. If the patient remained erect for 15–20 minutes, his blood pressure would drop and he would develop a shock-like syndrome so that he had to be main-

tained almost continuously in bed. The skin revealed stasis dermatitis, dependent rubor, and trophic changes over both lower extremities. There was a trace of pretibial edema. The results of the remainder of the general physical examination were within normal limits.

On neurological examination the patient knew his name but did not recall his address. He stated his age incorrectly. He felt that he was still at the other hospital and that it was probably the fall of 1933. He realized, however, that his memory was poor and had insight into the fact that he had been having difficulty naming objects over the past several days prior to admission. He was able to perform simple one-digit calculations. He could recall none of four objects after one minute. There was mild difficulty in naming familiar objects or parts of objects. The patient could obey spoken commands reasonably well but had more difficulty with written commands. He had difficulty in writing, manifested by holding the pen clumsily and by misspelling of words. His ability to write was clearly better than his comprehension of written material. There was minimal right-left confusion. The optic fundi revealed micro-aneurysms, deep punctate hemorrhages, and waxy exudates consistent with diabetic retinopathy. There was no papilledema. There was a dense right homonymous hemianopia. The right pupil was slightly larger than the left, but both reacted to light and on convergence. The rest of the cranial nerve functions were normal. There was a tendency for the right arm to drift downward and laterally when the patient's eyes were shut. There was severe wasting of all four extremities, especially distally. No fasciculations were observed. There was weakness of all four extremities in a distal distribution. Muscle tone was normal throughout. Fine movements were performed clumsily with the right hand. The deep tendon reflexes were absent throughout. The plantar responses were also absent. The patient showed an unsteady, wide-base gait. Sensory examination revealed that pinprick, light touch, and vibratory appreciation were impaired in a fading stocking distribution over both feet to the level of the knees. In addition, position was impaired in the toes of the right foot and the fingers of the right hand. Twopoint discrimination, tactile localization, and stereognosis were impaired in the right hand. The patient manifested extinction to touch over the right side of the body on double simultaneous stimulation.

During the first few days of his hospital stay, the patient developed congestive heart failure, pulmonary emboli, diabetic keto-acidosis and gastro-

intestinal bleeding, for all of which he was treated succesfully. Serial electrocardiograms were consistent with a diagnosis of pulmonary emboli. A bilateral superficial femoral vein ligation was performed. A lumbar puncture showed normal pressures, clear cerebrospinal fluid with 2 red blood cells, a sugar of 133 mg/100 ml (with a corresponding blood sugar of 200 mg/100 ml), and a protein of 256 mg/100 ml. The colloidal gold curve was flat. The patient's serum proteins were 5.2 g/100 cc, and there was a reversal of the albumin-globulin ratio. Serum electrophoresis was within normal limits. X-rays of the skull were also within normal limits. An electroencephalogram on the second hospital day was abnormal, with left postcentral preponderance due to the presence of theta waves at 4 to 7 per second. A repeat EEG about ten days later showed no essential change.

Because of the patient's condition, detailed examination of the higher functions was not possible until the fifth hospital day. At this time he was unable to name simple objects, written words, or letters. He was, however, able to name a circle, a triangle, and a square. There was no right-left confusion and no finger agnosia. The patient was able to follow verbal commands well. There was now some minimal weakness of the right lower extremity, above and beyond the weakness due to the patient's chronic sensorimotor polyneuropathy. On the 22nd hospital day the patient was still unable to name even simple letters, such as C or S. He could not read the words cat, dog, or his own first name. Using his left hand, he demonstrated that he could write much better than he could read. Although he could not read, he was able to copy letters as well as words without any difficulty. He was able to add simple 1 and 2 digit numbers in his head, but was unable to add the same numbers when they were written down on paper.

The patient was finally transferred to a nursing home on the 31st hospital day. The disability in reading was still present, although the medical problem had become well stabilized. During the next eight months, through the kindness of Dr. J. Litter, the patient's private physician, we were enabled to study the patient in detail, first at the nursing home, then at his own home, and finally (through the courtesy of Dr. William Timberlake) at the Lemuel Shattuck Hospital.

When the patient was first seen at the nursing home seven weeks after the acute episode, he demonstrated the following residual picture: The distal symmetrical sensorimotor polyneuritic signs were still present, as

well as the right hemianopia. Speech production and comprehension were normal. The recent memory deficits had, however, cleared completely (except for the special difficulties in topography to be mentioned below). The right-sided sensory loss which had appeared at the time of his acute episode had cleared (leaving only the symmetrical distal loss due to the polyneuropathy). Right-left orientation and finger-naming were normal, and he could perform mental arithmetic well. There were no disturbances of consciousness or abstract thought. He could spell words correctly and write them with either hand separately. Handwriting was normal but he wrote slowly. There were no constructional difficulties. Optokinetic nystagmus was present and grossly equal to both the right and left sides. These aspects of his clinical picture remained stable over the next few months.

He showed severe disturbances in spatial orientation and topography. Thus, he could not tell us where the nursing home or his own home were located and incorrectly answered questions about how to get to various points in the Boston area with which he had had extensive experience in the past. It should be stressed, however, that these disturbances cleared to normal in the next few weeks.

In the last months of his life, he developed some new symptoms consisting of vague pains over the right side of the body, especially the thorax, which were diffuse and difficult to characterize. They were increased by repetitive sensory stimulation. Although these raised the suspicion of an incipient thalamic syndrome and were repeatedly complained of, they never became agonizing.

In addition to the findings listed earlier, he showed certain disabilities which remained essentially stable over the next eight months and which were the object of special study. He complained of distortions of his visual field which we failed to be able to characterize. These, however, did not in any way affect his ability to copy visual forms or words correctly. He could neither read words aloud nor comprehend their meaning but could copy them correctly. He could not match words to appropriate pictures. He could read only some single letters; two-digit numbers, by contrast, were usually read correctly. Only one error was made in several months in the naming of objects, photographs, or line drawings. He could spell words presented to him orally and could comprehend words spelled to him orally. He could write spontaneoulsy and to dictation but could not read his own handwriting if it was shown to him later. Also, in strik-

ing contrast to his correct object identification was his difficulty in color identification, which was as persistent as his alexia. We will give in the next section a detailed account of the tests employed to study this color identification difficulty and the results obtained.

II. STUDIES OF COLOR IDENTIFICATION

Naming of Seen Colors

Colored sheets of paper were presented to the patient. He misnamed in almost all cases, and there was no consistency to his misnaming except that the response 'gray' or 'grayish' was the most common. His misnamings applied not only to colors in the narrow sense of the term but appeared also when he was presented with black, white, or gray.

Arbitrary Colored Objects:

Colored pictures of objects which are not usually of a fixed shade, e.g. neckties, crayons, dresses, blankets, or curtains, were then presented to the patient. His errors were similar to those produced with colored pieces of paper.

Distinctively Colored Objects or Pictures

Objects or pictures of objects which are usually of a certain color or are conventionally regarded as being of a certain color, e.g. apple, banana, telephone, milk, bricks, were presented to the patient. He failed in these as he had in the first two categories. These errors occurred although the patient could name the objects correctly and could, as shown below, tell what colors these objects normally were. Thus, the patient, shown a pad of writing paper, said it was tan; but when asked, said that writing paper was usually white. He insisted that this pad 'looked tan' to him. He misnamed the color of the red bricks in the fireplace in his own living room.

Verbal Memory for Colors of Objects

The patient was asked to state the usual colors of specified objects, e.g. apples, bananas, the sky, etc. He performed without error on this task.

Verbal Memory for Objects Having a Certain Color

The patient was asked to name the usual colors of objects named by the examiner. He performed without error on this task.

Comprehension of Names of Seen Colors (Matching of Seen Colors to a Name Given Verbally)

The patient was shown a group of colored objects or sheets of paper and was asked to select a specific color, e.g. "Show me the red sheet of paper." The patient failed in this task almost always. He would also fail in a simpler form of this task: being shown an object and asked, "Is this red?" he would answer at random.

Color-Matching

Several versions of this test were carried out and all were performed correctly. Thus, the patient, when given two rows of colored papers, would successfully match each paper in one row to the same color in the other row. In order to rule out that the patient was doing this task by means of brightness differences, a more complex form of the test was set up whereby the colors differed markedly in brightness and saturation. The patient performed without error in this task.

A still more complex task was set up in which the patient was given a pile of colored chips of papers and permitted to sort them. He correctly collected all of the items of a particular kind. Thus, he correctly sorted a group of chips of two slightly different shades of green into two separate piles.

Ordering Shades of One Color

The patient could correctly arrange a series of reds in order from bright red at one end to pink at the other.

Matching Seen Colors to Pictures of Objects

The patient was given a group of colored papers or crayons and a group of line drawings of objects. He correctly matched the colors to the objects, e.g. a yellow crayon to a banana.

Pseudo-Isochromatic Color Tests

The patient was given both the American Optical Company and the Ishihara pseudo-isochromatic tests of color vision. He performed both without error. The American Optical test was performed readily because it uses geometrical forms which the patient could name readily. On the

Ishihara test the patient either named the digits (as we have noted above the patient could read most two-digit numbers although he could not generally read two-letter pairs) or traced them out with his finger. On those parts of the Ishihara test using colored paths he traced these correctly. It should be noted that one of the examiners (NG), who is moderately red-green blind, performed very poorly on these tests, while the patient performed as well as the examiner (MF) with normal color vision. Despite this the patient's descriptions of the color in which the correct answer was printed were grossly inaccurate. Thus, one of the cards which shows a bright red 7 on a gray background was described as having a gray 7.

In addition to these tests of the patient's color identification ability, certain features of his performance on these tasks deserve to be mentioned here. One of these was the fact that the patient never replied with a simple "I don't know" to the demand for naming a color. Furthermore, when offered the correct name from a group, he would not accept it. In

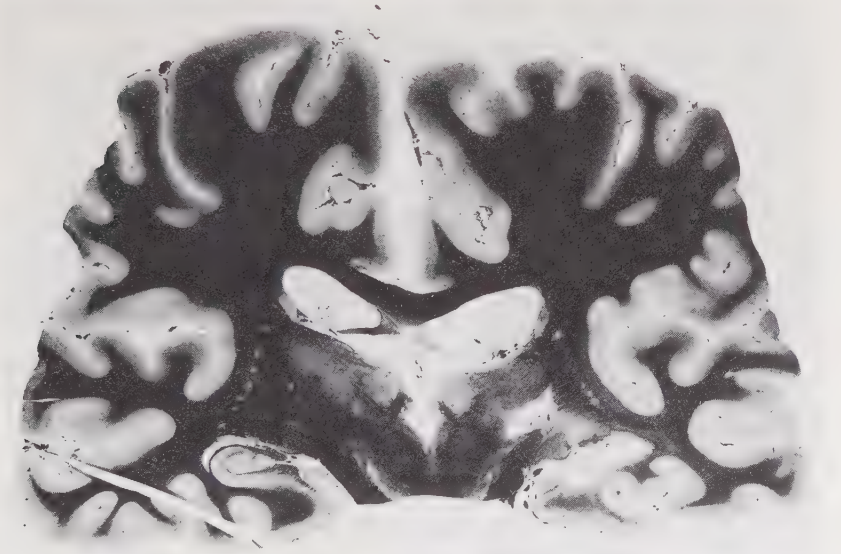

Fig. 1. In this section (2200) the total degeneration of the left fornix is seen as well as the cystic lesion in the left posterolateral-ventral nucleus of the thalamus. Destruction of the left hippocampus and enlargement of the temporal horn of the left lateral ventricle is also evident.

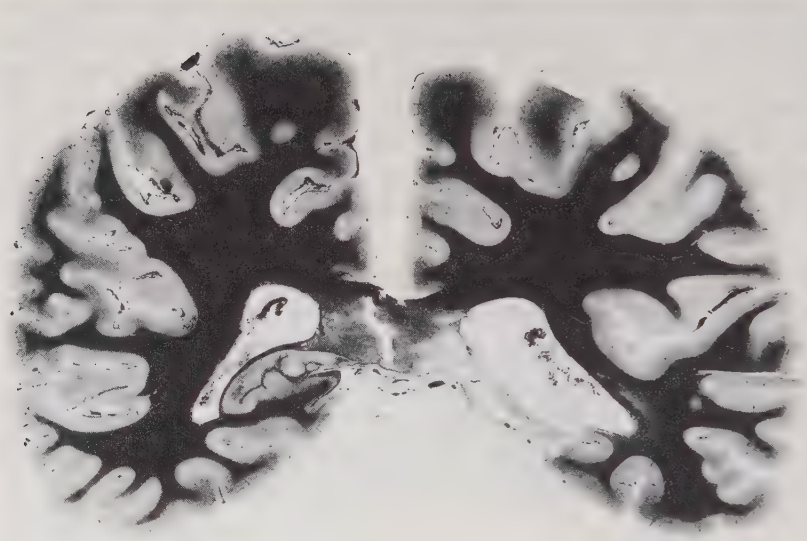

Fig. 2. Total infarction and degeneration of the splenium of the corpus callosum is seen (section 2500). The cavity in the center of the splenium is a result of artifact and was not seen in adjacent serial sections of this region. The gross destruction of the hippocampus on the left is also evident.

fact, if the patient had replied 'gray' and was told by the examiner that the correct answer was 'red', he would often say, "It may look red to you, doctor, but it looks gray to me" or "Well, maybe it's a reddish-gray." When pressed, he would almost always insist that the color 'looked' to him as he had named it. Sometimes he would explain his response by saying that his eyes weren't working well. On one occasion, shown a pad of writing paper, he said that it was tan. When we asked him what color writing paper normally was, he said, "It's usually white but this looks tan." When we told him that the paper was actually white, he suddenly said, "I don't understand what happened: it now looks white." We will return to this in the Comment section.

The patient only once made an error in sorting. Our usual technique for this task was to hand him a colored chip (without naming the color) and the patient was told to select all the chips which looked like the one demonstrated. This he did without error. His only faulty sorting occurred

on a variant of the task. We showed him a chip and said, "This is red. Now pick out all the red chips." He proceeded to form a pile which contained all the red chips but also all the chips of one of the two shades of green that were in the group. When asked what his pile contained, he picked up a red chip and said, "This is red." He then picked up one of the chips of the particular shade of green that he had selected and said, "This is another shade of red." We then had him match the chips from his pile (without our mentioning any color names) to the green chips of the shade he had not chosen. He carefully matched all of the shade of green he had chosen to these but did not select out any of the red ones. We will return to this curious result in our Comment section.

III. PATHOLOGICAL EXAMINATION

The patient died on Dec. 21, 1963, 15 months after the onset of his original difficulty, at the Lemuel Shattuck Hospital. The brain after removal was fixed in formaldehyde solution (formalin).

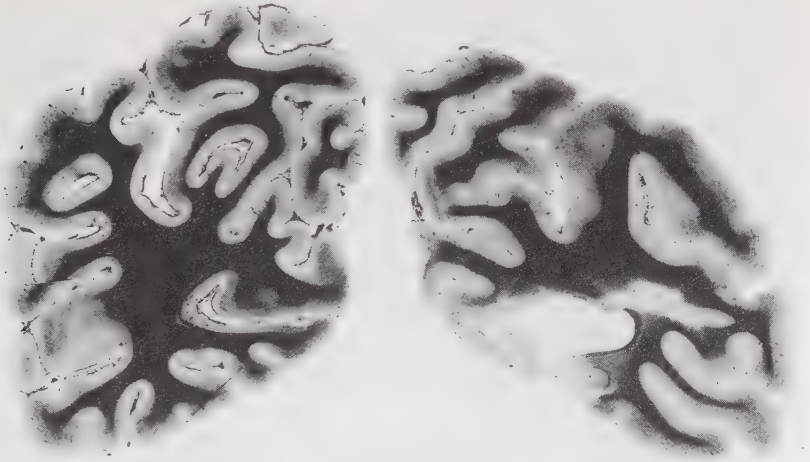

Fig. 3. The visual cortex on the right is intact and on the left is completely destroyed (section 3200). The optic radiations are also affected. The cystic cavity on the left communicates with the posterior horn on the same side.

On gross examination of the uncut brain fixed in formaldehyde solution, the only obvious finding was an old softening in the region of the left occipital pole.

The brain was embedded in celloidin, and sections were cut at 35μ intervals with alternate sections being stained for myelin and cells. Representative sections are shown in the figures.

The right hemisphere was normal except for secondary degenerations resulting from lesions in the left hemisphere. Figure 1 (section 2200) shows that the left hippocampus was infarcted with resulting complete degeneration of the left fornix. In addition, an infarct is seen in the posterolateral ventral nucleus of the thalamus. The corpus callosum is intact in this section. Figure 2 (section 2500) shows that the splenium is infarcted. Figure 3 (section 3200) shows the destruction of the left calcarine cortex by the infarct. No other lesions were found in the brain. It was concluded that the areas of infarction noted were all within the distribution of the left posterior cerebral artery.

IV. COMMENT

This 58-year-old man entered the hospital originally for investigation of a sensorimotor polyneuritis which was thought probably to be a complication of his diabetes. He exhibited severe postural hypotension which appeared after 15 to 20 minutes in the upright position and was thought to be a manifestation of the polyneuropathy. This may well have contributed to the acute episode of cerebral infarction from which the patient suffered. We will try to relate the lesions found at postmortem to the clinical picture. In essence, we found a long-standing infarction in the distribution of the left posterior cerebral artery. The left calcarine cortex, the splenium, and the left hippocampal region were extensively involved, and there was also an infarct in the ventroposterolateral (VPL) of the left thalamus.

Certain aspects of this clinical picture cleared during our period of observation. The early general recent memory deficit and the later difficulty in topographic memory were in this category, as well as the right-sided sensory loss. It seems reasonable that the right-sided sensory loss was a neighborhood effect of the thalamic infarct seen at postmortem. We had conjectured that during his life the patient's complaint (appearing late in

his course) of ill-defined vague pains over the right side of the body but sparing the face, and made worse by repeated stimulation, was an early thalamic syndrome, and this too may be attributable to the thalamic infarct which involved VPL but spared the trigeminal representation in ventroposteromedial nucleus (VPM). The early recent memory defect may have been only a nonspecific result of an acute episode or may have reflected bilateral ischemia of the medial temporal regions which produced transient but reversible damage on the right side, while leading to a permanent infarct on the left. The alternative possibility should, however, be kept in mind that an infarct of the medial temporal region on one side alone, particularly if that side is the dominant one, might itself be sufficient to cause transient recent memory disturbance despite complete normalcy of the nondominant side. The left medial temporal region might normally be most involved in verbal memory tasks since this is the side of the brain on which speech is localized. Thus, Meyer and Yates (1955) and Milner (1962) have shown that removal of the left temporal lobe back to 9 cm may produce a severe deficit in verbal memory, while a lesion of the right side does not have this effect. Both the findings of these earlier investigations and our own might be accounted for by assuming that the normal pathway for verbal memory is from left temporal speech areas to other areas of left temporal neocortex lying more anteriorly which project in turn to the medial temporal region. A left medial temporal lesion would thus produce a recent memory disturbance at least for verbal material. The impairment would probably be transient since impulses from the left speech region could eventually follow an alternative route. Instead of the probable normal route, speech area to the left anterior temporal neocortex to the left medial temporal region, the following route could be taken: speech area to the left anterior temporal neocortex to the right anterior temporal neocortex (via the anterior commissure) to the right medial temporal region. We carefully checked the anterior commissure in the sections of our patient since, in its role as the major commissural connection between the temporal lobes (with the exception of the first temporal gyrus), it would play an essential role in such a compensatory mechanism. The anterior commissure was intact in our patient. Because we cannot rule out that the patient had more widespread ischemia during the acute stage, particularly of the medial temporal regions bilaterally, we can do no more than suggest the possibility of this alternative mechanism.

The difficulty in topographic memory was less transient, since it was still present more than six weeks after onset. It did, however, disappear subsequently. This might be attributable to transient damage to the posterior parietal regions which were not far from his permanent lesions. It is, however, of interest that he did not at this time show a full Gerstmann's syndrome. Another possibility is that the difficulty in topographic memory was simply a part of his more widespread recent memory deficit and had merely cleared more slowly.

The more stable deficits included the right hemianopia which is clearly the result of the infarct of the left optic radiation and calcarine cortex. The persistent complaint of distortion of the visual field was of interest and is similar to phenomena reported by other patients with posterior lesions. We did not investigate this in detail and can only comment briefly on its possible significance. Whitteridge (1965), confirming earlier suggestions by Hubel and Wiesel, and Myers, has recently brought evidence to show that the midline of the visual field is represented by a thin strip of cortex, area 18s (or juxtastriate 18). This region, unlike the primary cortical representation of the other parts of the visual field, has callosal connections; and Whitteridge has hypothesized that these fibers, running in the splenium of the corpus callosum, may serve to maintain continuity of the two visual fields and also to insure that direction of line is preserved across the vertical meridian. A lesion of the fibers of the splenium which interrupted these fibres might lead to a disruption of this mechanism so that lines crossing the vertical meridian might be distorted. For this to be operative in our patient, it would be necessary to assume that a small midline strip of the right visual field would have had to be intact. While our fields showed no gross sparing, we cannot in retrospect rule out a mild degree of such sparing. We can only mention his mechanism here with the hope that this possibility may be looked into more closely in the future.

The features which were the major objects of our interest were the reading disorder and the difficulty in color identification. The reading disorder cannot be attributed to any elementary visual disturbance, such as the distortion complained of above, since the patient correctly copied the words he could not read and because there was no difficulty in describing complex nonverbal visual arrays. The patient clearly showed the classic syndrome of pure alexia without agraphia (Dejerine, 1892), many of whose aspects we have discussed elsewhere (Geschwind, 1962, 1965). The post-

mortem findings of infarction of the left calcarine cortex and of the splenium coincide with the findings in Dejerine's case which have repeatedly been confirmed.

Our primary reason for presenting this patient is that he provided an especially rich opportunity for prolonged study of the difficulty in color identification, the results of repeated study of which over several months we have summarized above. Inspection of these results shows that the patient's pattern of disabilities can be summed up in one simple formula: The patient failed in all tasks in which he was required to match the seen color with its spoken name. Thus, the patient failed to give the names of colors and failed to choose a color in response to its name. By contrast, he succeeded on all tasks where the matching was either purely verbal or purely nonverbal. Thus, he could give verbally the names of colors corresponding to named objects and vice versa. He could match seen colors to each other and to pictures of objects and could sort colors without error.

By no nonverbal criterion could our patient be shown to have any deficit in color vision. In fact, a laboratory animal testable only by nonverbal means, who scored as well as our patient did on the nonverbal tasks, would clearly be accepted as having normal color vision. Our patient's perfect performance on speudo-isochromatic tests of color vision reinforces this conclusion.

Our patient's one error on a special variant of the sorting test mentioned above is easily understood. We told the patient that a certain chip was red and to select all the other red ones. He picked up each chip and very slowly examined it. By specifying that we wanted 'red' chips, we forced him to name every chip rather than simply to match to the sample chip. By misnaming a chip of one shade of green, he made an error. This error, because of the forced introduction of verbal mediation, may be called a secondary sorting error. Pick (1931) referred to the possibility of this type of error. The importance of this type of error is that one must be very careful in testing for nonverbal matching or sorting not to introduce inadvertently forced verbal mediation which may lead to errors and confuse the examiner into believing that the patient had made a primary perceptual error. We suspect that some errors in sorting or matching described in the older literature may be on this basis.

One might describe the deficit as one of the naming of colors, but this might obscure the fact that the disturbance is a more wide-spread one

since, in fact, the patient also fails to comprehend color-names under circumstances specified above. It is interesting that a similar defect occurs frequently in normal children. Thus, at the time of this study, the same tests applied to our patient were given to the four-year-old son of one of us. He also named colors incorrectly and showed incorrect comprehension of color names yet correctly sorted and matched colors and traced out the correct responses on the Ishihara test. This is not uncommon in children of this age and usually clears within a couple of years, as has indeed been the case for this child.

Color-name aphasia, as it was called, was well recognized by the classic German neurologists (as summarized by Lange 1936). There was, however, some reluctance to accept the defect as one of color-naming alone. Some of the patients made errors in matching or sorting tasks. We have already alluded to some of the difficulties in the interpretation of such tasks. Another problem was the fact that these patients commonly refuse to accept the correct name, as our patient did, or to insist as he did that the color really looks to them like the color they have mentioned and not like the correct color named by the examiner. These facts led to an assertion that these patients were manifesting an 'agnosic' defect in addition to the naming defect. In order to consider this thesis, one must consider the anatomical lesions of these cases. Most typically the lesion, as first described by Dejerine (1892) for his case of alexia without agraphia, destroys the left calcarine cortex and the fibers of the splenium which carry the callosal fibers of the visual regions. Thus, the left visual cortex is destroyed and the right visual region has lost its commissural connections, so that it is cut off from the speech area in the left hemisphere. This visual-verbal disonnection explains both the reading deficit and the inability to name colors or comprehend their names. It is also evident that the patient will not accept the correct name if offered to him since, if his lesion is extensive enough, he will not be able to compare the name heard in the left hemisphere with the color seen in the right hemisphere. Nor can he describe his color-vision experiences correctly since the part giving the description, i.e. the speech area, is disconnected from the part having the visual experience. It is of interest that in this situation the patient does not deny knowing the name but produces confabulatory responses. It has been pointed out elsewhere extensively that many of the so-called 'agnosic' syndromes are actually produced by disconnections of primary sensory

regions from speech area, with the resultant confabulatory responses (Geschwind, 1965). A final problem is presented by the discrepancy between color-naming and object-naming. Adolf Meyer (1905) hinted at a possible mechanism. Thus, object-naming might be preserved despite the loss of reading in cases of pure alexia without agraphia because an object could arouse tactile associations further forward in the right hemisphere which could then get across the callosum over its intact portions anterior to the splenium. Written words lack tactile associations and, thus, would not reach the speech area via this roundabout route. Colors, like words, have verbal associations (their names) – but unlike objects, no tactile ones. The same explanation could account for the preserved reading of numbers in many cases of pure alexia, since numbers have powerful tactile associations as a result of the strong tendency to count on the fingers in childhood (Dejerine, 1892; Symonds, 1953). The role of tactile associations in the syndrome of pure alexia without agraphia has been discussed more fully elsewhere (Geschwind, 1965).

It is evident that our case effectively answers Critchley's (1965) skepticism concerning the existence of amnestic color aphasia and would seem to go against his alternative suggestion that these cases might be accounted for on the basis of the combination of minimal dysphasia with minimal perceptual deficit. Our patient was exhaustively examined to rule out any defect in color perception, and his normal speech and the presence of only one error in object naming over months of repeated examination rules out the possibility of a general anomic aphasia. Furthermore, since the combination of a readily observable mild anomia with a visual field defect is not rare, such striking disturbances of color-naming and comprehension should be common. In fact, they are rare and are not commonly seen in such cases with mild anomias and visual field deficits, as would be demanded by Critchley's hypothesis. The close association of color-name aphasia to pure alexia without agraphia and to a highly specific anatomical lesion clearly runs against the highly nonspecific hypothesis suggested by Critchley.

We do not wish to suggest, however, that all disturbances of color identification are clinically identical with the type discussed here or result from the mechanisms we have outlined. Some of the cases with distortions of color vision (rather than with difficulties of color-naming) presented by Critchley may well be the result of complex alterations in color percep-

tion. It is clearly of great theoretical importance not to confuse such cases with the type of problem we have discussed here. Thus, Kinsbourne and Warrington (1964) described a patient who had disturbances similar to those of our patient but also showed some difficulties in nonverbal matching, unlike our patient.

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NOTES

* From the Boston University Aphasia Research Center and from the departments of neurology of the Boston Veterans Administration Hospital and the Boston University Medical School.

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CHAPTER X

LANGUAGE-INDUCED EPILEPSY

PREFACE

This case, the first report of a patient with epilepsy induced by his own speech illustrates that in neurology, and in particular in the study of the higher functions, clinical observation is still an important tool. Nature carries out experiments in man that cannot be duplicated in animals and, even when we have no understanding of them, it is vital that they be recorded so that future investigators can study them. The syndrome of language-induced epilepsy remains a complete mystery, which is not explained by our current knowledge of the organization of language in the brain. It thus seems likely that it contains some clues as to some aspects of the neural organization of language of which we are as yet unaware.

It is interesting that there are cases of epilepsy induced by reading, writing, or speaking. Dr. Francis Forster of the University of Wisconsin has described a patient who developed seizures in response to the voice of a particular radio announcer. His patient however, responded to only one particular voice, while a patient with reading epilepsy usually has seizures induced by any written material in a given language. It is not clear whether there exists a form of epilepsy in which any heard voice can induce an attack.

LANGUAGE-INDUCED EPILEPSY*

1967

ABSTRACT. This to our knowledge is the first reported case of a so-called reflex epilepsy in which the seizures could be triggered by attempts to use three language modalities, i.e., reading, writing, and speaking, and is also the first case in which speaking has played a major role as a triggering stimulus.

This 47-year-old man who stuttered since childhood gave a history of seizures characterized by speech arrest, jerking movements of the jaw, and, on occasion, loss of consciousness. General physical, neurological, spinal fluid, and pneumoencephalographic examinations revealed no significant abnormalities. With the patient unmedicated seizures consisting of myoclonic movements of the palate followed by jerking of the jaw were repeatedly precipitated by certain language stimuli, ie, reading (aloud or to himself), writing, speaking, and whispering. Lip and tongue movements, however, and humming (without words) are ineffective. Although writing words was an effective stimulus, drawing of geometric forms was not. Speaking was sometimes not effective in producing a seizure until several seizures had been induced by reading. This phenomenon of 'sensitization' has not to our knowledge been described previously. Hyperventilation, intermittent photic and optokinetic stimulation were not provocative. The resting electroencephalogram was normal. During seizures (with retained consciousness) spike or polyspike and wave complexes occurred bilaterally, suggesting a seizure focus in the so-called centrencephalic system possibly at a pontine level. Ethosuximide and phenobarbital therapy produced a marked improvement in the clinical and EEG manifestations of this disorder.

The so-called reflex epilepsies, i.e., those in which the seizures can be precipitated by highly specific stimuli, continue to be of great interest to the neurologist. In discussing this form of epilepsy, we restrict the term 'reflex' to those instances in which a highly patterned stimulus regularly leads to a seizure by effects transmitted over nervous pathways. By contrast, in all other forms of epilepsy the seizure-provoking agent is presumed to be a local metabolic change within the focus itself or a change in some blood-borne substance capable of influencing the focus. The ready identification and, to some extent, possible manipulation of the specific stimulus hold forth the possibility of greater insights into the mechanisms of the epileptic process. In those instances where specific language stimuli appear to be the triggering mechanisms, the possibility of further understanding of the neurophysiology subsuming language itself makes a

thorough investigation of such epilepsies seem most rewarding. We have recently encountered a unique patient whose seizures could be triggered by three modalities of language, i.e., reading, writing, and speaking. To our knowledge this is the first case in which language in any of three modalities appeared to be a specific seizure-provoking stimulus and, in particular, the first in which speaking has played a major role in precipitating seizures. It is because of this unique specificity that this case is reported. A preliminary account of our findings appears in an earlier publication (Sherwin *et al.*, 1965).

I. REPORT OF A CASE

History

A 47-year-old man admitted Oct. 28, 1964, was referred by his own physician to the Neurology Service of the Boston Veterans Administration Hospital for evaluation of a seizure disorder. He had been a stutterer since early childhood. The past medical history was otherwise unremarkable. With the exception of the seizure problem, the patient considered himself always to have been in good health. Specifically, he was unaware of any perinatal complications and denied any serious illnesses of childhood, head trauma, or symptoms suggesting infection of the central nervous system. There was no family history of seizures, speech disturbance, or other neurological disorders. He had achieved only a tenth-grade education completed at age 16, which the patient attributed to the speech difficulties previously mentioned. At the time of admission, the patient complained of 'blackouts' which he had first noticed approximately 15 years before, when he was in his early 30s. Initially, the spells had occurred once or twice weekly, but there had been some diminution in frequency over the five years preceding admission. For a few months prior to admission, however, they had become more frequent once again. The spells were described as occurring without any warning. The patient would first notice jerking movements of the jaw, followed by some tremulousness of the hands. During this time there was speech arrest. On occasion, this was followed by complete loss of consciousness, but generalized convulsions never occurred. The patient's wife volunteered that some of these spells seemed to occur while the patient was reading, although many had occurred while he was not so engaged. Although the episodes themselves

were very disagreeable for the patient, he felt well immediately thereafter without any postictal deficit.

The general physical examination revealed a somewhat asthenic white male with a mild kyphoscoliosis. The rest of the general examination was unremarkable.

Neurological Examination

With the exception of the obvious stutter, the detailed neurological examination was completely within normal limits. Psychometric examination revealed a Wechsler full scale intelligence quotient of 82, placing the patient in the range of borderline deficiency.

Laboratory Findings

Routine examinations of the blood and urine were within normal limits. The cerebrospinal fluid was under normal pressure with a protein of 44 mg/100 ml and no cells. Plain X-ray films of the skull revealed no abnormality. A pneumoencephalogram disclosed a normal ventricular system and normal subarachnoid pathways.

Course

During the periods in which the patient was receiving no anticonvulsant medication, clinical seizures could repeatedly be precipitated by certain language stimuli. These included reading (aloud or to himself), speaking

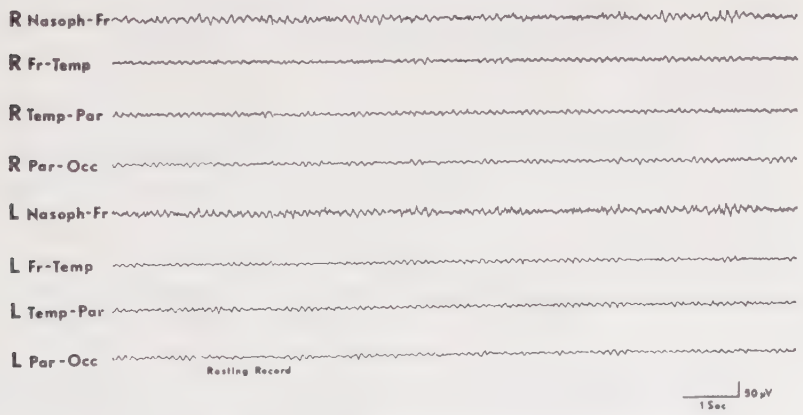

Fig. 1. Resting record, patient off anticonvulsant medication.

(aloud or whispered), and writing. The clinical seizures were noted to begin with speech arrest, although the patient might produce some guttural sounds. The arrest of speech was accompanied by myoclonic movements of the palate which were followed by jerking of the jaw. The spells were obviously most disagreeable to the patient, and he would resist the urging of the examiner to continue the activity after a seizure had been provoked. During the spell the patient remained conscious and was able to respond to verbal commands. Other provocative techniques which included hyperventilation, as well as intermittent photic and optokinetic stimulation, were all without effect. Although speaking and whispering words were both effective in precipitating seizures, repetitive lip, tongue, and jaw movements and humming of a tune without words were not effective stimuli. Although writing was noted to be an effective stimulus, drawing of geometric forms failed to precipitate seizures. Of the three language modalities reading was the most powerful stimulus. We noted that it was often necessary to provoke one seizure by reading before writing or speaking could be effective in precipitating any further seizures. However, once the patient had been 'sensitized' to speaking or writing, he then remained highly susceptible to these forms of stimulation during the remainder of the test period. When sensitization was maximal, a seizure might be provoked by repeating a single word spoken by the examiner. In some

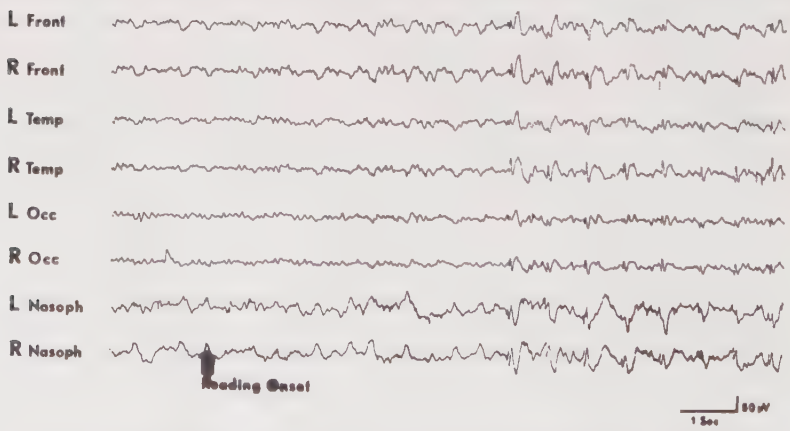

Fig. 2. Seizure induced by reading; monopolar recording to both ears interconnected. Excess background slowing probably related to several seizures precipitated just prior to this record.

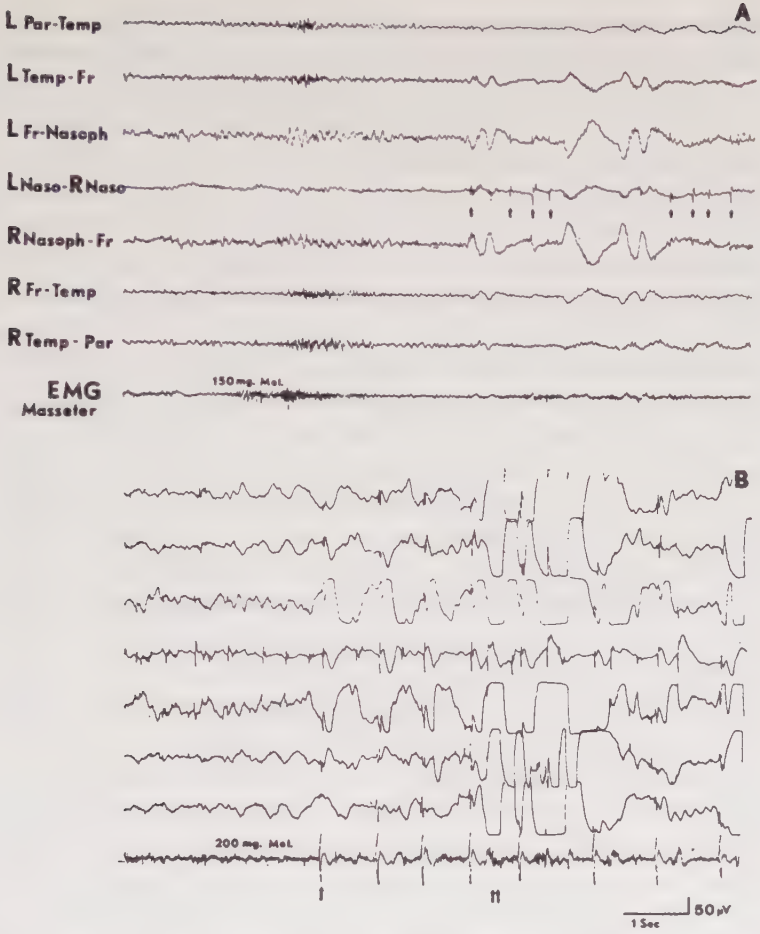

Fig. 3. Metrazol activated EEG. (*A*) following intravenous injection of 175 mg, electromyographical potentials recorded from palate (*arrows*); (*B*) electromyographical potentials from masseter (*single arrow*), onset of jerking of head and eye blinking (*double arrow*).

instances the seizure activity would begin before the patient had even completed the repetition of the word.

Electroencephalographic Examination

Several EEGs were recorded both from the convexity and from naso-

pharyngeal leads. The resting record (Figure 1) revealed a predominance of well modulated 7 to 9 cycles per second activity bilaterally. Recordings made during sleep revealed no paroxysmal activity. Intermittent photic and opticokinetic stimulation carried out during EEG recordings also failed to produce any paroxysmal activity. During clinical seizures induced by the previously mentioned provocative stimuli, bilaterally symmetrical spike or polyspike and wave complexes, at maximum amplitude in the frontal derivations, appeared in the record (Figure 2). A simultaneously recorded electromyogram from the masseter muscle (not shown) indicated that the cortical discharge did not bear a one-to-one relationship to the muscle potentials. In an effort to determine, without using one of these 'triggering' stimuli, the origin of the initial electrical discharge a pentylenetetrazol- (Metrazol) activated EEG was performed. Following the injection of only 175 mg of pentylenetetrazol, a clinical seizure was provoked. This seizure started with myoclonic jerking of the palate, followed by jerking of the jaw and finally by intermittent contractions of the orbicularis oculi. The patient remained conscious, was able to follow verbal commands, but was unable to speak. The first recorded electrical manifestation of this seizure was rhythmic electromyographical activity picked up from the palate by the nasopharyngeal electrodes (Figure 3, *A*). This was then followed by rhythmic electromyographical activity in the masseter muscles, with only rare spike and polyspike wave complexes appearing over the convexity (Figure 3, *B*). Though distorted by excessively prominent artefacts, this record also showed that a one-to-one relationship between the cortical and electromyographical potentials did not exist. The patient was treated with ethosuximide and phenobarbital with a marked improvement in both EEG and clinical manifestations of the seizure disorder. He has been seen several times in follow-up continues to do well on this regimen.

II. COMMENT

The patient presents several interesting features. On admission he was thought to be a case of reading epilepsy. Shortly after anticonvulsant medication was withdrawn, one of the house staff reported that the patient was having repeated spontaneous seizures. Careful examination, however, soon revealed that the seizures were not really spontaneous but occurred only when he attempted to speak. As long as he lay quietly, he had only

a very occasional mild attack, while attempts at speech, reading, or writing invariably produced a seizure. This observation is in keeping with the increasingly frequent recognition and identification of specific, provocative 'triggers' in apparently 'spontaneous' seizures (Servit, 1963).

A second point of great interest was the increased ease of precipitating seizures after at least one seizure had been induced, usually by reading. This feature of sensitization appears to be a most unusal one, for a search of the available clinical literature failed to reveal any discussion of this curious phenomenon. Whether it is present to some extent in all forms of epilepsy or only in certain forms of reflex epilepsy is not known. Since each individual seizure occurs only in response to a specific stimulus, the situation is different from that in status epilepticus which it may superficially resemble. In his study of after-burst activity, Burns (1955) has suggested that sustained cortical discharge may be due to a chronic differential (surface positive) repolarization of an aggregate of neuronal tissue. Such a state of heightened excitability may in fact be analogous to the dominant focus produced by cortical anodal polarization (Rusinov, 1953) or by tetanizing stimulation of the cortex (Roitbak, 1959). These latter studies indicate that alterations in the regional steady potential may increase the excitability of an area of cortex so that it responds to certain stimulus modalities to which before sensitization it was unresponsive.

It would seem possible, then, that a first seizure induced in our patient by reading might alter the steady potential in the focus and thus facilitate the triggering or repetitive after discharge by related stimuli in a pattern determined by the conditioning process.

To be an effective trigger such a stimulus would have to arrive at the focus during the period that such a regional alteration in the steady potential persists, viz, usually only for a few minutes (Rusinov, 1953). It must be acknowledged that such speculation as to the physiologic substrate of this phenomenon of sensitization is purely theoretical and not supported by any direct evidence in this case. However, though the exact mechanism of this phenomenon remains obscure, it seems worthy of further study since in many cases of reflex epilepsy certain stimuli which might be effective as triggers would be missed should they only be examined for during 'interictal' periods, without first subjecting the patient to a sensitization procedure.

The EEG findings were also of some interest. The fast transients, first

seen in the nasopharyngeal leads, were soon found to be 'muscle artefacts' resulting from palatal movements. The EEG changes found in this case appear to be essentially the same as those reported in primary reading epilepsy (Bickford *et al.*, 1956; Baxter and Bailey, 1961; Stevens, 1957). This pattern of activity suggests that the electrical focus resides in some portion of the so-called centrencephalon, perhaps at a pontine level.

The most interesting feature of this patient and the one which prompted this report is the ease with which speaking or whispering could induce seizures although the use of the same musculature in nonverbal tasks had no effect. This case illustrates the great specificity of the stimulus. In evaluating the possible mechanisms involved in this disorder, certain interpretations may readily be rejected. It is extremely difficult to accept the view that these are movement-induced seizures since nonverbal movements of the cranial musculature and limb musculature were both ineffective in inducing seizures, while movement used for the purpose of producing language readily provoked the fits (Asbury and Prensky, 1965). This is not to dispute the existence of movement-induced epilepsy (Gowers, 1901; Falconer *et al.*, 1963; Symonds, 1959) but only to point out that in the case described movements *per se* were not the effective stimulus. Proprioceptive feedback could similarly not be the provoking stimulus since this would fail to account for the verbal-nonverbal distinction (Bickford, 1954; Alajouanine *et al.*, 1959). Furthermore, if proprioception were the essential stimulus, it is difficult to see why in the case of reading epilepsy reported by Stevens (1957) different languages showed such marked variation in their ability to trigger seizures. Concentration could hardly be a significant factor since even the most trivial verbal activity was sufficient to produce seizures when the patient was sensitized. Similarly, the view that combinations of such less specific factors might be the responsible triggers (Critchley *et al.*, 1959–1960) is not supported by a critical review of the reported cases of reading and writing epilepsy (Sherwin, 1966). Indeed, it is difficult to accept any conclusion other than that the specific seizure-provoking aspect of the stimulus was its verbal character. Further, it is not necessary to be surprised that so specific a stimulus may trigger a seizure, in the light of the fact that one of the most common activities of the nervous system is the linking together of highly specific stimuli. So common is this activity that it is perhaps almost surprising that highly specific reflex epilepsies are not more frequently encountered.

It should be noted that seizures precipitated by speaking may be more common than has been appreciated and that, in particular, many cases of reading epilepsy may also show this disturbance, especially if 'sensitization' by reading is first used as a provocative technique. Bickford (written and oral communication to the authors, March 12) has noted occasional provocation by speech in some of his reading epileptics.

We were unable to find a recorded case of epilepsy precipitated by hearing spoken language. It is, of course, difficult to conceive how a patient with such an ictal disturbance would get along since, while it is easy to avoid writing, reading, and speaking to a considerable extent, it is extremely difficult to avoid hearing spoken language. It would therefore seem that such a patient would be bombarded by seizure-provoking stimuli. It is possible, however, that this might eventually result in 'desensitization' to this form of stimulation so that one might mistakenly assume that the patient was immune to it. A clinical example of such auditory desensitization may be found in Forster's case of musicogenic epilepsy (Forster *et al.*, 1965). In any case, one should not exclude the possibility that 'heard-word' epilepsy exists without first attempting possible sensitizing procedures. We did not, for example, carefully study our patient to see whether spoken words could provoke seizures in him after a long series of attacks precipitated by reading, writing, or speaking.

One curious aspect of nearly all the cases of primary language epilepsy is that the EEG is nonfocal and that one does not find focal EEG changes in the speech areas of the dominant hemisphere. It differs, therefore, from many cases of reflex epilepsy in which cortical foci are the rule. It appears that there may be two major types of reflex epilepsy. One is the variety in which a simple stereotyped stimulus (e.g. viewing a safety pin in the classic case of Mitchell *et al.*, 1954) is seizure provoking. In such cases cortical foci are the rule. The other group comprises those forms of epilepsy in which a highly specific class of stimuli is effective, although the actual individual stimulus may be different in each instance. Thus, in language-induced epilepsy completely different words will be effective on different occasions. In such a case we find no cortical focus. This distinction may well point to some highly significant factors in physiological organization, but it is not possible even to speculate about these at this time.

A final word is appropriate on the patient's 'life-long' speech difficulty. Its onset in childhood, the characteristic clonic blocking with grimacing

aggravated by any deliberate effort to speak unhaltingly, and its subjectively disagreeable nature all would seem to identify the disorder as stuttering as opposed to cluttering (Luchsinger and Arnold, 1966). That the spontaneous speech difficulty is not identical with the clinical seizure is apparent for the following reasons. The stuttering spontaneous speech was not accompanied by either the clinical manifestation of the seizure, i.e., palatal myoclonus, jaw jerking, or by EEG changes. Secondly, when seizures as manifested by EEG changes occurred, the above clinical features were always apparent. The lack of EEG abnormality during the spontaneous stuttering speech further distinguishes the dysphemia from the 'cluttering-syndrome'. (Landolt and Luchsinger, 1954). Lastly, the differential response of the seizures and the spontaneous stuttering to anticonvulsant medication further served to identify them as distinct phenomena. None of the other patients with reading epilepsy so far reported has been stated to be a stutterer so that perhaps this association in our case is only coincidental. Our case, however, differs from the others in the ready provocation of seizures by speaking. One could speculate that perhaps the stuttering was instrumental in linking speech production to the patient's epileptic process. Conversely, it is conceivable that the stuttering is, in fact, the result of the seizures. Thus, in our patient at the onset of a seizure induced by speaking, his stutter would worsen. This is highly speculative, of course; and even if true in this case, this mechanism must be a rare cause of stuttering since EEG studies have not in the past revealed such abnormalities in stutterers.

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CHAPTER XI

THE VARIETIES OF NAMING ERRORS*

1967

ABSTRACT. Four syndromes are discussed in which there occurs failure of naming on confrontation. The first is the anomia of posterior aphasia-producing lesions. The second is a much more delimited disturbance produced by lesions which isolate certain sensory regions from the speech area (probably from the left angular gyrus in particular) and which may be best characterized as failure to match certain sensory stimuli to their spoken names. The third syndrome is that of non-aphasic misnaming which occurs in disorders which diffusely involve the brain. The fourth is anomia occurring as a manifestation of hysteria or malingering. The clinical syndromes are outlined as well as the distinguishing features of the anomia in each type. Some of the theoretical problems raised by each form of anomia are also discussed.

In this paper I shall be discussing anomia in the sense of the inability of the patient to name things shown to him, what can be called failure of naming on confrontation. This task makes up so large a part of the testing of aphasia that it deserves attention. It is sometimes even asserted that failure in this task occurs in all aphasics. While as a rough statement this may be true, it deserves modification before it can be accepted without reservation. Thus there are certainly aphasics in whom naming on confrontation is very good relative to the patient's abnormal spontaneous speech production, even if rarely fully normal. This is particularly true in some cases of Broca's aphasia, and Jakobson (1964) has made much of this in his description of polar types of linguistic disturbance in aphasia.

I would stress also that we are speaking of naming in the narrower sense and not of word-finding in the flow of speech. The reasons for the failure to find words in one situation while they can be found in another have not yet been adequately studied and will not be discussed here. Instead I shall simply confine myself to discussing four distinguishable varieties of failure of confrontation naming which may have some theoretical importance in considerations of aphasia, and are of significance in clinical diagnosis.

The localization of anomia has often been a disputed point. In classical writings there were two opposing views, one which asserted that anomia resulted from a lesion of the left parietotemporal region while the other

claimed that it was the result of diffuse disorder of the brain. The proponents of each localization had supporting data for their point of view. Gloning *et al.* (1963) dealt with this question simply by accepting both types of localization. As I will try to show, this problem can be at least partially resolved by the demonstration that local and diffuse localization respectively correspond to different types of anomia.

One must of course be sure that the failure to name an object is not the result of failure of perception. Thus if a patient calls a 'pencil' a 'fork' it is conceivable that this could be the result of perceptual distortion. A test which is often used to rule out perceptual error is to offer the patient names and to see if he chooses the correct one. This test can, however, serve to exclude perceptual disturbance but cannot be used to prove that it is present. Thus, if the patient always selects the correct name from those offered to him he must in each instance have recognized the object. Failure to accept the name might, however, be due not to incorrect perception of the object but to incomprehension of the name. Other tasks must be used to insure the correctness of perception. Correct pantomine of the use of the object proves correct perception. Similarly, matching tasks may be used. If a patient calls a 'pencil' a 'fork' but invariably matches a pencil to another pencil or a picture of a pencil, one must rule out perceptual difficulty. Similarly, if the patient is shown an object and misnames it but then draws it correctly, we must again exclude perceptual disturbance.

The classic form of anomia is characterized by difficulty in naming on confrontation, while the patient usually but not always accepts the correct name. The patient may also show by many of the other maneuvers listed above that he recognizes the object. He may simply fail to produce the name, or he may produce an incorrect name or a circumlocutory description. Almost invariably, when confrontation naming is impaired the patient's spontaneous language production is disturbed so that while speech is fluent, it may contain many paraphasias, circumlocutions or 'empty' words (such as 'thing', 'place', 'these'). The word-finding difficulty in spontaneous speech is not confined to the names of physical stimuli but also involves abstract nouns and adjectives and other parts of speech (Wepman *et al.*, 1956; Howes, 1964). When in addition to these features the patient's comprehension and repetition of speech are normal we have the classical syndrome of anomic aphasia, the lesion of which usually

lies in the left temporo-parietal region (the angular gyrus). If disturbances of either comprehension or repetition, or of both are present in association with the above picture of fluent, paraphasic speech, the localizations are somewhat different although in all instances the lesions lie behind the Rolandic fissure.

In this first group the anomic difficulty tends to be a rather general one. It involves all modalities and all types of stimuli. The intensity of the disturbance is not, however, necessarily equal for all stimuli, as has recently been shown in careful studies by Goodglass *et al.* (1966).

In contrast to the above type of anomia we have another more restricted form, of which I will cite two examples, both of which I have presented elsewhere (Geschwind, 1965) although I did not in my earlier discussion bring out some of the differences between this type and the classical form of anomia discussed in the preceding paragraphs.

An example of this variety of anomia was seen in the patient of Geschwind and Kaplan (1962) who had a proven infarction of the corpus callosum sparing only the splenium. He had no difficulty in naming objects visually and speech was normal. When blindfolded he misnamed objects held in the left hand. The misnamings were bizarre, e.g., 'eraser' for a ring, 'balloon' for a watch, 'book of matches' for a padlock, 'an elastic' for a nail, 'piece of paper' for a screwdriver. Furthermore, he did not accept the correct name when it was offered to him. That there was no perceptual failure could, however, be proven by the fact that after feeling an object with the left hand while blindfolded he could, using the same hand, pick the object by touch alone out of a bag or select it with eyes open from a group. When blindfolded he would use the object correctly with the left hand simultaneously misnaming it. In addition he could afterwards draw the object correctly with the left hand. It is clear that perception was intact and that this must be a naming defect.

It might be objected that one could explain this disturbance as the result of a combination of a minimal sensory defect in the left hand with minimal general naming disturbance. This is similar to the explanation advanced by Critchley (1966) to account for cases of color-naming difficulty. This type of explanation can be shown, however, to be inadequate to account for the naming difficulty of our patient for objects held in the left hand. The patient had definite sensory loss in the *right* hand as manifested by position, vibration, and two-point testing. In the *left* hand he could be

shown by nonverbal testing to have normal sensation. Yet he correctly named objects felt with the right hand but not those held in the left. This fact clearly rules out the explanation of sensory loss plus minimal naming difficulty since naming of objects held in the right hand should then have been more seriously involved than that of objects held in the left hand. I suspect that this type of explanation will rarely if ever be justified in any case. It is difficult to believe that a combination of two borderline disturbances could produce so dramatic and delimited a syndrome as this patient demonstrated. This case similarly cannot be accounted for by dementia or uncooperativeness since these would fail to explain the unilaterality of the disturbance.

A second example of this variety of anomia illustrates an even more delimited syndrome. This is the color-naming defect which accompanies some cases of pure alexia without agraphia. Geschwind and Fusillo (1966) have recently described such a case in detail. Consistent results were found on repeated careful testing over a nine-month period. This patient had a right hemianopia. He could not read, but he named objects normally (only one error in object-naming was made in all the testing sessions). He misnamed colors, but his misnaming always consisted of other color-words. The responses were not consistent in that the same red might on one occasion be called 'gray', on another 'green'. The answer 'gray' was, however, more common than any of the other color-words. The patient failed to select the correct answer from a group. Furthermore, he failed in the task of selecting a named color from a group presented to him. On the other hand, perceptual deficit was ruled out by the fact that the patient matched hues correctly despite wide differences in brightness or saturation, that he could sort colored chips correctly, even sorting into separate piles chips of slightly different shades of the same hue. He correctly matched colored pieces of paper to uncolored pictures of objects. Finally he performed normally on two pseudo-isochromatic tests of color vision, the Ishihara and the American Optical Company tests. Loss of memory for color-names was ruled out by the fact that he correctly answered questions such as, "What is the color of a banana?" Kinsbourne and Warrington (1964) in an earlier publication described a similar, although not identical, case.

It is obvious that this too is a naming disturbance. Critchley (1966) has suggested, as was noted above, the possibility that this disorder is not a

specific entity "but rather a very mild disorder of visual perception coupled with a minimal dysphasia." This explanation is difficult to accept in the face of the powerful evidences of normal color perception I have cited above, and does not account readily for the highly delimited character of the syndrome. Furthermore, many patients with the combination of an obvious mild anomia and a hemianopia do not show dramatic difficulty in color naming, although one would expect this to be a common effect of this combination if Critchley were correct. Furthermore, since some degree of color-blindness is presented in about 7% of males, one would expect this percentage of mildly aphasic patients to show dramatic color-naming difficulties. Yet color-naming disturbances are rare and are most likely to occur in association with another rare syndrome, that of pure alexia without agraphia which has a characteristic pathological picture (Geschwind, 1962).

Both of the cases described above have come to anatomical examination, the brains having been cut in whole-brain sections, with alternate sections stained for myelin and cells. In the case of Geschwind and Kaplan the corpus callosum was infarcted and the splenium was spared. In the case of Geschwind and Fusillo the left visual cortex and the splenium were destroyed. The tactile naming difficulty in the left hand in the first case was interpreted as the result of disconnection of the right somesthetic cortex from the speech area, and the second case was interpreted as the result of disconnection of the right visual cortex from the speech area. Since I have discussed these explanations in detail elsewhere (Geschwind, 1965) I will return to our central interest at this moment, the character of the anomia in these cases.

Difficulties in naming, predominantly in a single modality, have been mentioned by many authors although these have frequently been treated as agnosias. Arguments against this interpretation have been summarized in Geschwind (1965) and will not be repeated here. Two excellent recent examples of dissociation between modalities are seen in the case of Ettlinger and Wyke (1961), who named better visually than by touch, and in the case of Spreen, Benton, and Van Allen, who cited two patients with predominantly visual and three with predominantly tactile anomia. Kaplan and Geschwind (unpublished) observed one patient with a predominantly tactile naming disturbance.

Let us look at the features of this second form of anomia which dis-

tinguish it from the classical form. In the first place the second type of anomia is remarkably delimited, being confined to the tactile modality in the left half of the body in the first case and to color-naming in the second. The classical type of anomia is more global in its effect. In the second place spontaneous speech was normal in both of these cases and neither patient showed word-finding difficulty except on confrontation. Thus the second patient showed no difficulty in finding color words except when confronted with a color. This contrasts with the classical form of anomia in which even mild confrontation naming difficulty is almost always accompanied by word-finding difficulty in spontaneous speech as manifested by hesitation, particularly in finding substantive words (as against 'grammatical' words), by an increased tendency to use 'empty' words, by circumlocution, or by production of paraphasias.

A third difference is in the nature of the errors. The classic anomic may fail to name or may produce verbal paraphasias, combinatory paraphasias (e.g. 'ring-key' for 'key-ring'), phonemic paraphasias, or even grossly neologistic errors. Patients with the second variety of anomia usually produce no combinatory or phonemic paraphasias or neologisms. This is not the result of differential severity of naming disturbance since classic anomics even when mildly impaired may produce phonemic paraphasias while the cases of the second type of anomia discussed above produced no phonemic paraphasias despite gross naming failure in the affected spheres.

The errors of our patient with the tactile naming disturbance in the left hand were in general 'wildly' incorrect and not errors in sphere (i.e., related by sound or meaning) such as are often produced by the classic anomic. It may be argued that in the case of color-naming deficit the patient did produce errors in kind since he always produced the name of some color. This, however, overlooks the fact that it was the method of testing which led to this result. We would ask the patient, "What color is this?" and he would therefore confine his reply to color-names. It is obvious that our first patient also used the information from the question "What do you feel in your left hand?" to give names of small palpable objects. This is a much larger class than that of colors and permitted therefore of apparently more random answers.

A fourth difference, one of major importance, is the fact that the patients with the second type of anomia showed not only disturbance in naming but also what might at first be thought of as comprehension disturbance.

Thus, these patients failed to choose the correct name from a group. The second patient could not select a named color from a group. This special type of comprehension disturbance was not recognized classically since it was taken as evidence of 'agnosic' difficulty. We have, however, already shown that these failures were not due to perceptual disturbance, but we can also see that they are not failures of comprehension in the usual sense. Thus these patients both perfectly understood the names of objects and colors in such sentences as "Tell me the name of something red" or "Are roses green?" A more accurate description would be to say that the patients could not match the sensory stimulus to the spoken name. The classic anomic is like the second variety of anomic in failing to produce the name when presented with the stimulus, but differs from the second variety in usually being able to choose the correct stimulus from a group when presented with the spoken name.

Another difference lies in the patient's response to his deficit. The classic anomic may admit to not knowing the name. On the other hand he may show various forms of denial. He may insist that he never knew the name, or that he has always called it by the term which he had just used, even when this is clearly incorrect. He may assert that he really knows the word, but I suspect that this 'tip-of-the tongue' phenomenon is at least as often mistaken in the patient as it is in normals.

The second group of patients almost never admit to not knowing the correct name but will almost always produce a response. When pressed as to the reasons for the discrepancy between his response and the one designated by the examiner as the correct one, the patient may assert that he hasn't done this kind of thing in the past, or that things 'look different' to him from the way they look to the examiner. In short the classic anomic acts as if his vocabulary and that of the examiner differ; the patient with the second type of anomia may act as if his perception and that of the examiner differ although perception can be shown to be intact by nonverbal methods.

We can summarize the two groups as follows. The classic anomic has difficulty in naming on confrontation which more or less involves all types of material and is more or less equal for all sensory modalities. The inadequacies of naming may consist of failure to produce a response, verbal paraphasias which are generally related by sound or meaning, phonemic paraphasias, or circumlocutory descriptions. There is usually

difficulty in spontaneous speech, manifested by word-finding pauses or any of the other difficulties listed above. The patient can usually choose the correct name if offered to him by the examiner and can select a named object from a group. The patient ascribes his errors to differences in knowledge or usage of words. The lesions in these cases are in the dominant angular gyrus region.

Contrasted with this is the second type of anomia, best characterized as an *inability to match a stimulus to its spoken name*, i.e., the patient can neither produce the name when presented with the stimulus nor choose the correct stimulus when given a name. The difficulty is confined to a single modality, and even to a single class within that modality, and may be confined to one-half of visual space or one side of the body. Circumlocutory errors or phonemic paraphasias occur uncommonly if at all. The errors consist primarily of verbal paraphasias which are not related to the correct word by sound and are related only in the most general way by meaning. Spontaneous speech is normal even for the categories which are specially impaired in confrontation naming. The patient cannot usually choose the correct name of the item when it is offered to him; nor can he select a named item correctly from a group. Comprehension deficit in the usual sense, however, is ruled out because the patient shows normal understanding of the same words when he is obliged to give purely verbal responses. Perceptual deficit is ruled out by the patient's ability to carry out the various tests listed earlier in the paper. The patient may ascribe his errors to differences in perception between himself and the examiner. The lesions are those which produce disconnections from the speech area.

It is of some importance to consider the differences between these groups of anomic disturbances. To call one disturbance aphasic and the other perceptual or agnosic runs counter to the demonstrated fact that perception can be shown to be intact even in those patients suffering from the second group of disturbances who assert that their difficulty is perceptual. A point of great theoretical importance which arises out of a careful consideration of the differences between the two groups is that one can safely reject the thesis that classical anomic aphasia is the result of the accumulation of large numbers of separate naming disturbances. Since the isolated naming disturbances are likely to be of the second variety a large number of these appearing together in the same patient would produce a picture quite different from classical anomia as any consideration of the dis-

cussion above will clearly show. Thus phonemic paraphasic errors may be seen in classical anomia which is also associated with difficulties in spontaneous speech but without comprehension deficit; in all of these ways it differs from the isolated naming disturbances. Classical anomia appears therefore to differ not only in degree but also in kind from the isolated naming disturbances.

It seems clear, however, that occasionally one must encounter patients with classical anomia who also have the admixture of one or more isolated naming disturbances thus leading to an apparent selective impairment of certain types of naming. Unfortunately there is little documentation as to whether such patients show the specialized type of comprehension disturbance specified above for the second group. Thus in the studies of Goodglass *et al.* (1966) cited above it may be that the increased difficulty in certain naming tasks in classical anomics may reflect admixtures of 'disconnection' anomias. If this is the case then one should find difficulties in comprehension of the specialized type I have described in these categories above. It will be important to investigate this further in the future.

How can one account theoretically for these two varieties of anomia? The second type, i.e., the 'disconnection' anomia, appears to be more readily accounted for than does classical anomia. Thus if a lesion cuts off most of the connections between the cortical representation of a given sensory modality and the speech area, it is easy to see that the patient will name incorrectly since the sensory information will fail to reach the speech area. It is also easy to see that the patient will have difficulty matching a name spoken by the examiner to the stimulus since there is no path from the speech area to the association cortex of the modality in question. The patient will make no errors in matching *within* the affected modality, nor will he have any troubles in purely verbal tasks. Spontaneous speech would be normal. The patient will often refer to his errors *as if* they were due to perceptual difficulty since the speech area which is giving the account is indeed not receiving the perceptual information. As stated above, the lesion by disconnecting the speech area from some given sensory region prevents the matching of a sensory stimulus to a speech stimulus, regardless of the order of presentation.

Classic anomia implies, however, a lesion in a more complicated system. This system can be conceived of as one which receives the sensory information and finds the spoken word appropriate to it. It is a system which

may be thought of as containing the rules for finding a word given some of its attributes. Obviously it uses information from all modalities since it can 'find' a word when presented with the actual object in one or more modalities or on the other hand it can find the word when given its attributes in verbal form. As may have been noticed, this system is referred to as finding the *spoken* word corresponding to a set of attributes. It is my guess that it would act primarily to find the auditory form of words since it is this form which is first learned. Finding the written word would therefore be secondary to finding the auditory form.

It is reasonable that the region involved in the activity of finding a word given some of its attributes in one or more modalities is the angular gyrus since, as I have suggested elsewhere (Geschwind, 1964, 1965), this region is probably involved in the formation of associations between the various non-limbic sensory modalities (vision, somesthesis, audition). It is also easy to understand why damage to such a region should leave the grammatical structure of spoken language so well preserved. The 'small' grammatical words depend little if at all for their evocation on attributes in different sensory modalities. More generally grammatical structure, e.g., rules of word order and their transformations are almost certainly not dependent on such intermodal interactions but probably develop, like the small grammatical words, almost entirely within the auditory system.

It is reasonable that such a set of rules would be formulated in a large region of gray matter which is probably more or less equipotential. It also must be so designed that it is easier to find the attributes given the word than to find the word given the attributes since patients with lesions in this system comprehend the word more readily than they can find it. The system is therefore in some ways like a punch-card system. It also seems reasonable, however, to assume that in the most extensive cases of destruction of the area involved in this activity significant comprehension deficit would occur although it is not seen in partial destruction. I have made the speculation elsewhere (Geschwind, 1965) that perhaps these more extensive cases correspond to the 'syndrome of the isolated speech area' but I will not go further into this problem at this point. In any case, it seems clear that we need further study of the anatomical connections of the angular gyrus region and further investigation of the classical anomias in order to learn the rules by which words are encoded in this

region. It would also seem reasonable that the delimited anomias ultimately depend on disconnections of a particular sensory modality from the left angular gyrus.

Having discussed these two types of anomia, each with localizable lesions (in the left angular gyrus or in connections to the left angular gyrus respectively), let us consider now a third variety. As was pointed out earlier, classically there was a disagreement as to the localization of anomic aphasia. One group attributed it to focal disease in the angular gyrus region while others claimed that diffuse lesions of the brain were necessary. As I have already noted, both groups were in fact partially correct. It is clear, however, that what was not realized classically was the fact that the anomias in the two type of syndromes were different. Edwin Weinstein and his coworkers (e.g., Weinstein and Keller, 1964) have made the most significant contribution in presenting the characteristics of anomia which may occur in acute diffuse disease of the brain. It is quite clear that many neurologists have overlooked this syndrome. On the other hand, it is necessary to avoid the reverse error, i.e., that of mistaking focal aphasic disturbance for this condition of 'non-aphasic misnaming'.

Non-aphasic misnaming typically occurs in disorders which diffusively involve the nervous system, especially when the disturbance comes on fairly rapidly. It is thus very common in head injuries, subarachnoid hemorrhages, rapidly increasing intracranial pressure, and in toxic or metabolic disorders such as intoxications, withdrawal syndromes, uremia or congestive heart failure. Thus in my experience the syndrome of non-aphasic misnaming has only rarely been seen on the Aphasia Unit in the Boston Veterans Administration Hospital, is occasionally seen elsewhere on the Neurology Service, but is more often seen in the medical or neurosurgical wards. By contrast, misnaming in the aphasic whether in classic anomic aphasia or as part of one of the other syndromes resulting from post-rolandic lesions can occur in chronic, stable form over months or years in patients having focal unilateral lesions without evidence of widespread diffuse involvement.

Non-aphasic misnaming is perhaps a poor term for the syndrome as a whole since it highlights only one feature of the disorder. Depending on the particular variation seen in any one case, the examiner may be deceived into thinking that the patient is aphasic, or that he is suffering from a disturbance of memory or of perception, or indeed that the patient

is deliberately being facetious (Weinstein terms this 'ludic' behavior, and it clearly corresponds in good part to the classical moria or Witzelsucht). This is illustrated in the sort of reply that one often gets from such patients when the question, "What do I do?" is made by the examiner. A typical response is "You're someone who asks a lot of questions" or "You're an investigator of some kind." Characteristically the errors tend to 'propagate'. Thus the patient being asked where he is may say "In a bus", and may then say that the examining physician is the driver, that those around him are passengers and that the bed he is in is used by the driver for resting. It is usually obvious once a sequence of questions is asked that ordinary aphasic misnaming is readily ruled out. Thus in aphasic misnaming there is no tendency to 'propagation' although perseveration, i.e., repetition of the same incorrect word, occurs frequently. On the other hand perseveration can also occur in non-aphasic misnaming and may thus present a difficulty of interpretation. In aphasic misnaming the problem rarely arises that one has to consider the possibilities of memory loss, perceptual difficulty, or jocularity since these are easily ruled out. It is much more difficult to exclude these in non-aphasic misnaming since it often appears impossible to obtain the patients' cooperation. The aphasic with misnaming who has no comprehension deficit usually accepts correction by the examiner but in non-aphasic misnaming this is often difficult or impossible to achieve.

The 'connected' or 'propagated' character of the errors may show up particularly in relation to the hospital and the patient's illness. He may call the hospital a 'hotel', the doctors 'bell boys', the nurses 'chambermaids', and will not accept correction. One man dying with terminal cancer said that the building in which he was located was a 'slaughter house' where 'they hang up bodies' and that the examiner was a 'grinder'. He denied specifically that it was a hospital. This example of what Weinstein calls 'tragic ludic behavior' (equivalent to 'gallows humor') illustrates another characteristic of the syndrome, i.e., that it is often possible to demonstrate that the patient in fact knows the correct answers since his errors are so clearly motivated. This is well illustrated by Weinstein's patient who asserted that she was in the 'Mount Cyanide Rest Home'. She could have produced this pun only with the knowledge that she was in fact in the Mount Sinai Hospital. This motivated misnaming is not a feature of aphasic anomia in which the patient will usually show no in-

creased difficulty in talking about his illness or the hospital and may even perform at his best when the examiner turns to these topics.

One feature which often characterizes non-aphasic misnaming is that spontaneous speech is usually (but not invariably) normal despite gross errors in naming. In aphasic anomia errors in confrontation naming are almost always accompanied by a disturbance in spontaneous speech in which word-finding pauses, empty phrases, paraphasias and circumlocutions appear.

Some of the errors in non-aphasic misnaming may appear to be more 'aphasic' than the 'motivated' or 'propagated' misnamings. In some instances rather bizarre errors occur, misnamings apparently totally unrelated by sound or meaning, but also not occurring in an obvious connected context. The differential diagnosis of these may be quite difficult. Thus while it is true that classic aphasic anomia usually leads to errors which are related in sound or meaning to the correct word it is clearly the case that such bizarre errors do occasionally occur, particularly in that group of patients with the syndrome of the 'isolated speech area' which Goldstein (1917) discussed and which I have commented on elsewhere (Geschwind, 1965). In these cases, however, the patient suffers from a gross comprehension disturbance, often with echolalic repetition, and can thus be easily distinguished from cases of non-aphasic misnaming.[1]

It is not possible at this time to give a more detailed account of the pathogenesis of non-aphasic misnaming. While it typically occurs in situations in which there is diffuse involvement of the nervous system this does not necessarily rule out that it is some focal aspect of the diffuse involvement which is really important. It is true that a single stable unilateral lesion has not been shown to produce this syndrome. It is possible, however, that stable bilaterally symmetrical lesions in some particular area are necessary. Certainly many aspects of this syndrome are reminiscent of some of the features of chronic bilateral frontal disease, but it would not be possible with assurance to equate non-aphasic misnaming with frontal disease. Furthermore, it is possible that this syndrome may occur transiently with acute unilateral disease in some specific focus and disappears in time, but this would be difficult to disentangle from the more widespread effects of acute lesions. In addition the place of involvement may not be cortical but may be subcortical as Weinstein himself has suggested. Unfortunately our knowledge of well-defined chronic behavio-

ral syndromes from non-cortical lesions is confined to a small number of disturbances such as Korsakow's syndrome or akinetic mutism. A further possibility is that the name-finding system of which we spoke earlier in this paper and which is located in the angular gyrus region may be very readily disturbed by diffuse disease because it depends so heavily on multiple connections from many regions.

The diagnosis of non-aphasic misnaming presents many difficulties. While one may see a relatively 'pure' example, e.g., the patient who is intoxicated, the differential is much more difficult when there is concomitant focal damage. The problem is compounded by the fact that the syndrome of non-aphasic misnaming may be superimposed on a mild focal anomic disturbance or on a Korsakow's syndrome. Examination is particularly difficult in such cases because the cooperation of the patient is often extremely difficult to obtain. Thus, we have seen cases in which once the non-aphasic misnaming had cleared, it became obvious that the patient was suffering from a classical Korsakow's syndrome. In other cases with clearing of the picture of non-aphasic misnaming we have observed a mild but definite aphasic anomia with an appropriate left posterior Rolandic lesion. This syndrome remains a major problem for students of the neurology of behavior and has been generally neglected except in the pioneering work of Weinstein.

We have now presented three situations in which misnaming may occur – classical anomic aphasia, those disconnection syndromes in which matching of a stimulus with its spoken name is impaired, and the syndrome of non-aphasic misnaming. We now turn to a fourth group of disturbances which includes hysterical and malingered anomias. In these cases the patient often fails to find the names of very simple objects presented to him. He usually shows great apparent concern over the failure. If the examiner insists that he is sure the patient can find the word, he often receives a response of annoyance or a protest that it is impossible to go on. These responses can also occur but are much less likely in aphasic anomia. The clue to the diagnosis in these cases is that despite a gross global defect of confrontation naming in all modalities, the patient's spontaneous speech is normal. Thus, one patient whom I examined would fail to name as common an object as a pencil presented either visually or tactually and yet could tell us with perfect fluency in her spontaneous speech that her husband was an electronics engineer who was working

on the design of digital computers. The anomia may be the only obvious abnormality or may occur against the background of a more outspoken hysterical pseudo-dementia, e.g., an outspoken Ganser syndrome. This 'functional' misnaming syndrome is rare but failure to recognize it may have serious consequences.

NOTES

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[1] Dr. Ennio De Renzi has pointed out to me that the syndrome of non-aphasic misnaming is similar in many ways to that described by continental psychiatrists as disordered language in mental confusion. He points out that a further useful criterion is that aphasics will generally have more difficulty in naming uncommon as against common objects while the patient with non-aphasic misnaming may paradoxically do better in the naming of uncommon objects. This same association could of course be seen in patients with hysterical or malingered naming disturbances.

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CHAPTER XII

WERNICKE'S CONTRIBUTION TO THE STUDY OF APHASIA*

1967

In this symposium we are honoring the memory of Kurt Goldstein. It is appropriate in this setting to discuss the contribution of Carl Wernicke to the study of aphasia, a field which concerned Goldstein throughout his life. Like Wernicke, Goldstein was born in a small town in Upper Silesia and received his medical education in Breslau, the provincial capital. During Goldstein's period as a medical student, Wernicke held the chair of neurology and psychiatry in the medical school and it was from Wernicke that Goldstein first learned about aphasia. The influence of this period on Goldstein was great and I shall comment upon this later.

I have in an earlier paper written about Wernicke's life and the lives of some of his most important pupils, as well as the general effect of the Breslau school on the history of aphasia (Geschwind, 1966). In this paper I will not review this earlier material extensively but rather will treat in detail Wernicke's contribution to the study of aphasia, in particular his classic work, 'Der aphasische Symptomencomplex' published in 1874.

In order to put this publication in perspective, let us briefly consider its background. Wernicke received his medical degree in 1870 at the age of 22 from the University of Breslau. He worked for six months in the ophthalmology clinic under Forster who was probably the first to recognize the young man's exceptional talent. He then went off as an assistant to a surgeon in the Franco-Prussian war. On his return to Breslau he took up a post as assistant in the psychiatric clinic. Carl Weigert, the developer of the stain for myelin that led to such important advances in neuroanatomy, was a fellow assistant. During this time Fritsch and Hitzig reported their important discoveries on the localised excitability of the cerebral cortex of man. Wernicke spent six months with great neuroanatomist Meynert in Vienna and this was probably the most important influence of his life. Having returned from Vienna he soon afterwards published 'Der aphasische Symptomencomplex' which was completed in May, 1974, less than four years after his graduation and after only three years of training in

neurology. He was only 26 years old in the month that he completed the manuscript.

The major influence on this work was the short period with Meynert who influenced not only Wernicke but also many others, including Flechsig, the pioneer of developmental studies of the brain, the great anatomist Forel and Freud. Meynert was one of the very first to be concerned with the relationship of anatomy to function, an interest that he transmitted to Wernicke. Thus, in his early work Meynert correlated the increased size of certain portions of the motor pathways in the brain stem and spinal cord of the kangaroo with the great preponderance of the hind-limbs in that animal, and showed the contrasting pattern in the bat with its predominance of the forelimbs, thus also establishing himself as one of the pioneers in comparative neuroanatomy. He correlated development with order of myelination thus laying the ground for Flechsig's later work. He brought order into the fiber systems of the brain by distinguishing the projection pathways connecting the cortex to the lower centers, the association pathways connecting different regions of the same hemisphere, and the commissural systems joining corresponding points in the two hemispheres. He pioneered the study of cortical cytoarchitecture by being the first to point out regional differences of cellular architecture in the cortex. He was the first to point out that the interior parts of the brain had motor functions while the posterior parts were sensory in nature. In reviewing these accomplishments it is easy to see why it was written of him that 'Erst seit Meynert ist das Gehirn beseelt' ('Only since the work of Meynert has the brain been given life'). As I will point out later, Meynert made a specific contribution to the study of aphasia that has generally been overlooked and which influenced Wernicke's thoughts on aphasia.

In the short space of 13 years between the publications of Broca in 1861 and Wernicke in 1874 interest in aphasia had grown rapidly. Bastian and Hughlings Jackson were already active in England, as were also a large number of French and German writers, and the literature of the subject was already sizeable. Wernicke's contribution to this growing literature was entitled 'Der aphasische Symptomencomplex: eine psychologische Studie auf anatomischer Basis' ('The Symptom-Complex of Aphasia: a Psychological Study on an Anatomical Basis'). In the very opening of the paper he points out that he will apply Meynert's teachings

to the problem of language and aphasia. Following this brief introduction we come to the first section in which Wernicke immediately makes clear his theoretical position.

He begins this section by pointing out that the doctrine of the equopotentiality of the brain which had been advocated by the great French physiologist Flourens had been recognized as untenable for some time but that it was equally incorrect to substitute for it the view of the phrenologists who arbitrarily assigned individual territories of the cortex to mental functions (as arbitrarily defined by the terminology of ordinary speech). Wernicke thus rejected the approach to cerebral function which denies all localization, but he equally strongly rejected the approach which was to appear in more sophisticated form in other writings on aphasia, i.e., the one which assumed that for every specifiable language function there was a particular piece of cortex assigned to that function, the approach of such authors as Henschen and above all Kleist (who, although a student of Wernicke's, eventually adopted a theoretical view of quite different nature from his).

In rejecting these extreme approaches which we may label as the holistic and mosaicist respectively, Wernicke offers his alternative: "...only the most elementary psychic functions can be assigned to defined areas of the cortex..." and as examples of such elementary functions he cites visual, olfactory, and tactile perceptions in the central projection areas of the respective sensory nerves. "Everything which goes beyond these simplest functions, the association of different impressions into a concept, thinking, consciousness, is an achievement of the fiber tracts which connect the different regions of the cortex to each other, the so-called association system of Meynert. We will see later to what extent these more complicated achievements are even now amenable to examination." This then is the underlying thesis: the higher mental functions are not properties of localized regions of the brain, but are constructed out of association of different regions, and it is the anatomical connections of these regions which make possible these associations.

Wernicke now turns to developing his ideas in detail. It is a characteristic of all nervous tissue, he writes, to preserve the effects of stimulation beyond the duration of the stimulus itself. In peripheral nerves this effect is brief and accounts for some of the phenomena of after-images, but in the cortex these residua of previous excitations may remain for long

periods and it is these residua for which the name 'memory-images' is suggested.

Basing himself on what then were recent physiological and anatomical observations, Wernicke points out that the parts of the brain lying anterior to the central fissure of Rolando are motor regions, while the remaining portions of the hemispheres are sensory in function. Thus the memory images of movements must lie in the frontal lobe, those of sensations in the appropriate regions of the other lobes.

He now turns to the problem of volitional movement. This is distinguished from reflex movement in two ways: (1) It does not occur in immediate response to an outside stimulus but can result from the memory images of former perceptions which need not be aroused in all instances by an external stimulus. Hence, although volitional movements differ from reflex movements, they have an essential unity with them. (2) The volitional movement is adapted more closely to its goal. It is, so to speak, 'preformed' being built up of memory images of previous movements.

Volitional movements are thus also reffex movements but are distinguished by their dependence on remembered stimuli arising within the cortex which arouse remembered movements stored elsewhere in the cortex. As the number of sensory and motor images increases, the more associational possibilities exist, the greater becomes the variety of movements, and the greater becomes the choice of movements which may be available in any given situation, the exact selection depending on all the factors involved at any one time. Thus writes Wernicke in closing Section I, "The only correct scientific definition of free will agrees most closely with this mechanical concept of the origin of spontaneous movement."

In Section II Wernicke turns to the specific problem of aphasia. He comments that although Broca's view as to the importance of the posterior end of the third frontal convolution for language had encountered considerable opposition, the publication of so many cases in agreement with his ideas, and the lack of effective counter views had led to continuing acceptance of Broca's position. In the interim, however, Meynert had published a most important work in which he demonstrated that the central projection of the auditory nerve lay in the cortex of the Sylvian fissure, and to this cortical region he assigned the designation of *Klangfeld* (sound-field). He supported his view by numerous autopsy

findings in aphasics in which either the insular region or the adjoining parts of the Sylvian fissure were altered. Many academic physicians had adopted this view since it corresponded to the vaguely felt need to bring hearing into association with the process of speech and because it helped to account for the fact that most cases of aphasia in which Broca's area was intact had changes in the region specified by Meynert, i.e. in the temporal operculum and adjoining regions.

I might comment here that the significance of Meynert's contribution to the study of aphasia has generally been overlooked. In the discussions of the history of aphasia that I have so far consulted, he is mentioned only as Wernicke's teacher. Nielsen (1946) in his bibliography cites two works by Meynert on aphasia published in 1866 but does not refer to these in the text. It is important to appreciate that it was apparently he who first convincingly showed that aphasia could occur in temporal lobe lesions. It was Wernicke's function to complete the cycle started by Meynert by showing how this fact achieved meaning in terms of Meynert's own studies of the brain, and to point out the important fact that this new localization corresponded to a clinical picture different from that seen in lesions of Broca's area.

Wernicke now develops his central argument. The frontal lobe was already known at that time to be motor in function. Hitzig (cited in a footnote on p. 7) had shown that stimulation of the lower end of the Rolandic cortex in monkeys led to movements of mouth and tongue. Broca's area must be the center for the representation (i.e., memory-images) of the movements of this region, while the first temporal gyrus lying at the central projection of the auditory nerve must be the center for the images of sounds. The fibers between these regions, which Wernicke thought to run in the insula, must be the mediators of the psychic reflex arc between the heard and spoken word. It should be pointed out that Wernicke (1908) was later to modify his view on the path between the two cortical speech areas and to accept von Monakow's view that it ran not in the insula but in the arcuate fasciculus, arching from the posterior temporal region around the back end of the Sylvian fissure and then running forward in the white matter of the parietal lobe until it reached the posterior inferior frontal region.

Wernicke now gives his analysis of the types of aphasia to be expected from various lesions. Peripheral lesions of the auditory pathway, not in-

volving the first temporal gyrus, lead to deafness but not to disturbances in speech production in the adult, who already has a rich store of earlier acquired sound images which he can reproduce at will. But if peripheral deafness occurs before there is a consolidated store of sound images, the child remains mute since he has acquired no store of sound images with which to arouse movements of speech. Sound thus has a special relationship to speech which is shared by no other sense organ. As Wernicke notes mutism occurring in a deaf child is a common and easily understood disorder, but the rare situation of a child endowed with normal hearing who cannot learn to speak rightfully deserves the name of congenital aphasia.

Wernicke enlarges his discussion of deaf-mutism to include an intriguing suggestion whose speculative character he is quick to admit. Of what significance, he asks, is unilateral deafness in childhood? This would lead to deaf-mutism only if the right ear were affected and only the left hemisphere capable of acquiring language, which seems unlikely. But perhaps right-sided deafness leads the child to develop the right hemisphere more for language than the left since he is now depending on his left ear. Could, he asks, such a process account for the existence of people with speech in the right hemisphere? This argument of Wernicke's would have seemed totally untenable a few years ago since the representation of each ear in both cerebral hemispheres (unknown at the time Wernicke was writing his paper) would seem to invalidate it. However, the recent emphasis on the Broadbent technique (Kimura, 1961) has again highlighted the more intimate relationship of each ear to the opposite hemisphere and perhaps warrants re-consideration of this speculation of Wernicke's.

Wernicke continued his analysis of aphasia to discuss the effects of a lesion of the first temporal gyrus, which has come to be called Wernicke's area in his honor by later authors.[1] The sound images of the names of objects will be lost. From this region arise the fibers which run from the auditory to the motor region, but these are now destroyed. In addition the association fibers from this region by which other sensory images are aroused will be destroyed. "The patient is thus neither capable of repeating nor understanding the spoken word." The representations of speech movements are retained since these were learned earlier and thus "the capacity to speak is preserved but with some limitation. For in the

usual act of speaking the sound image always appears unconsciously to be innervated simultaneously (as is indeed readily understandable from the process of development of speech), to be so to speak simultaneously hallucinated, and thereby to exercise a continuing correction on the course of the movement representations. The deaf person... still makes full use of this corrective mechanism." The patient is thus also aphasic in speech as a result of the loss of this unconscious correction exerted by the sound image. "The errors consist in the ready confusion of words... . He uses the same words now correctly, now incorrectly...." In the milder cases it is names of objects and actions which are most affected but the conjunctions and other words necessary for sentence structure are preserved although these can be lost in more severe cases. But in all cases two cardinal features persist – the patient has many words, and he lacks comprehension of spoken language.

In order to understand Wernicke's next comment we must try to place ourselves in the position of his readers. Wernicke was describing what he thought to be a new clinical syndrome. Although he was not cognizant of the fact, Bastian (1869) and Schmidt (1871) had already described aphasias with comprehension disturbances, but these papers had gone unnoticed and it was Wernicke who was the first to succeed in calling receptive aphasias to the attention of the medical world. Wernicke had, however, gone further than these authors in linking incomprehension of language to a type of speech disturbance different from that of Broca's aphasia, i.e., to what we would now speak of as a fluent paraphasic disturbance. Finally he had linked this new syndrome to a localization different from that of the classical Broca's aphasia.

Wernicke writes, "That cases with such manifestations have not been observed, or at least not published, until now, rests not only on the rarity of such cases but also on the fact that thoroughly experienced and intelligent physicians regard this condition as a confusional state – as I myself have had the opportunity to experience. For the psychiatrically trained man who knows the clinical forms of confusional states, the diagnosis has not the least difficulty." It is perhaps unfortunate that nearly a hundred years after the publication of Wernicke's paper the error he pointed out is still a common one, and patients with fluent paraphasic syndromes are still often misdiagnosed as confused or psychotic.

Wernicke continues to list some further features of his new syndrome.

A large potential vocabulary is a cardinal feature of this form, in contrast to the situation in Broca's aphasia. There is no hemiplegia, in contrast again to Broca's aphasia. "Agraphia is present. Writing is a conscious movement which is learned with intimate dependence on the sound and is always executed under its direction. Self-observation teaches us (and clinical experience agrees with this) thus between the movement of writing and the concept there is no direct connection such as must be assumed between the speech movement and the concept." Wernicke now argues, however, that the educated reader will not be alexic since he has learned to understand the written word without a rendering into spoken form although he will read aloud aphasically. The uneducated by contrast who always reads aloud will be alexic. This supposition would probably not be accepted today. Wernicke goes on to point out that with other lesions in the visual regions, agraphia and alexia alone could occur.

A comment is appropriate here on this syndrome of fluent paraphasic speech and disturbance in writing with comprehension difficulty for spoken and written language which came to be known as Wernicke's aphasia. It should be noted that Wernicke did not use the term 'word-deafness' which was introduced by later writers. Furthermore a certain number of authors state incorrectly that Wernicke claimed that a lesion of the first temporal gyrus produced the syndrome of pure word-deafness, i.e., difficulty in comprehension of speech out of all proportion to disturbances in the other modalities. Wernicke did not describe this syndrome nor did he even envisage its existence in this paper. It was not until Lichtheim (1885) that the theoretical possibility of this syndrome was suggested, and only later was it described clinically by Liepmann who was one of Wernicke's assistants in Breslau (Liepmann, 1898). Furthermore, Wernicke did not conceive of the syndrome he described as being a composite one, i.e., he did not argue that four separate lesions were necessary to produce the full syndrome, but asserted instead that as a result of the way in which language is learned and used all modalities would be affected by a disturbance of the auditory memory images.

Wernicke now considered the effect of a lesion involving the fibers connecting the sound field to the area of representations of movement but leaving each one of these regions intact. Wernicke called the resulting disturbance conduction aphasia (Leitungsaphasie). The patient understands everything, and can speak freely, but this speech is altered in the

same way as in the form of aphasia just described resulting from a lesion of the auditory field. For, although the sound images are preserved in conduction aphasia they cannot exert an influence on the choice of words. The partient can judge, however, whether his own productions are correct or incorrect and shows concern over his errors. In pure form, the patient will show in speech a great similarity with the sensory aphasic, but his comprehension is intact. This form of aphasia will show disturbances in reading and writing depending on how these were learned in childhood.

Wernicke omits any mention of the feature that was later to be regarded as the characteristic component of conduction aphasia, i.e., the disturbance of repetition in the face of fluent, paraphasic speech and good comprehension. Goldstein (1927) was later to regard this form of aphasia as the most truly aphasic of all types. Accordingly, he gave it the name 'central aphasia' but specifically equated it to the conduction aphasia of other authors.

Wernicke, like Goldstein later, thought that this form of aphasia resulted from a lesion of the insula, since he thought that the pathway from the posterior to the anterior speech regions travelled by way of the insula. Later authors, however, believed that the lesion for it lay in the arcuate fasciculus, which was shown to be the actual pathway between these regions, a view which, as we have already noted, Wernicke (1908) came to accept.

Wernicke lists finally the effects of destruction of Broca's area. The patient understands everything but is mute or employs only a few simple words, which he uses mostly for the designation of objects. Most previously described cases of aphasia belonged to this group. Wernicke thought that agraphia would not result unless the patient had been accustomed to speak to himself while writing. The patient will not understand written language unless he has always read aloud.

Finally he turns to lesions in the descending pathways from the frontal lobe. These he assumes could produce aphasia, but may not do so since the descending fibers may be rearranged so that individual parts of the speech act might be impaired, thus leading to dysarthria. Wernicke shows no knowledge in this analysis of the role of callosal connections or of the bilateral innervation of the speech musculature. His failure to appreciate these facts made him believe that a *complete* lesion of one cerebral peduncle could lead to aphasia. His pupil Bonhoeffer (1914) was to add

refinement to this analysis by considering the role of callosal connections and bilateral innervation.

Wernicke closes this analysis by assuming that in many cases the patient improves because of eventual substitution by the other hemisphere, a view that has remained a source of unsettled controversy even today.

Wernicke now discusses the problem of those deaf-mutes who by means of very highly complicated special teaching techniques learn to produce speech. He argues that in such patients the auditory soundfield must play less of a role than it does in the normal acquisition of speech. Such patients could therefore become aphasic but their lesions would involve different parts of the brain from those which would have to be damaged in normals. In particular, lesions of the first temporal gyrus might well be ineffective in producing aphasia. He urges the importance of studying post-mortem material from deaf-mute aphasics. We see here again Wernicke's emphasis on the fact that aphasia is produced not by lesions of fixed language areas but rather by damage to those areas which in any individual have been used in acquiring speech.

Wernickè now turns to a discussion of 'Asymbolie', a term introduced by Finkelnburg. He argues that loss of recognition of an object results from the loss of the optic, tactile, and auditory components which go to make up the concept. The patient so affected would differ from the aphasic in that he would indeed fail to understand the significance of objects he has perceived. Wernicke admits that current anatomical knowledge is insufficient to specify the locus of the lesions. We see here a discussion of what later was to be called agnosia, to the study of which Lissauer (1889), one of Wernicke's assistants, made significant contributions.

Section III of the paper gives histories and clinical findings of ten patients. The first case was that of Suzanne Adam, a 59-year-old lady who showed very poor comprehension and repetition, fluent, mildly paraphasic speech, alexia and agraphia, without gross neurological findings otherwise. She showed marked improvement, except for her agraphia. She did not come to post-mortem examination. However, writes Wernicke, this case recalled to him another earlier case, who had not been so well studied because, he says, at the time he saw her he had not yet worked out a correct analysis of the symptom complex of aphasia. This was the case of Susanne Rother, a 75-year-old lady who had been regarded when first admitted as suffering from aphasia with confusion. Her speech, while not so plenti-

ful as that of the first cited case, was fluent and paraphasic. (It should be noted that the word paraphasia was never used by Wernicke at this time and came into literature later. He speaks instead of misused and distorted words.)

She had severe comprehension deficit which made some people think she was deaf. At post-mortem the Sylvian branch of the left middle cerebral artery was occluded. There was softening of the 1st temporal gyrus, and this was the only focal lesion found in the hemispheres.

It is on this case 2, admittedly studied inadequately, that the Wernicke argument rests since the remainder of the cases discussed did not show the cardinal syndrome described in the paper, that of fluent paraphasic speech with comprehension difficulty. Case 8 (Louise Funke), however, came to post-mortem after the paper was completed and her findings were given in a brief appendix to the paper. In life she had shown the use of only one word 'ja' and understood nothing. At post-mortem a major branch of the Sylvian branch of the middle cerebral artery was thrombosed. There was a large Sylvian infarct which included both Broca's area and the 1st temporal gyrus. Wernicke writes, "... in both cases (this one and case 2) the first temporal gyrus and its anastomosis with the second were involved. Both suffered from sensory aphasia. Could this agreement be the result of a coincidence?"

On this meager anatomical evidence his argument rested. Like Broca's first case it was a slim reed on which to found so thoroughgoing a theory of aphasia. Other authors have claimed that none of Wernicke's cases supported his assertions, but this seems difficult to accept since the clinical and post-mortem findings of case 2 and case 8 were clearly consistent with his thesis although they were not sufficient to prove it. Henry Head (1926) dismissed Wernicke's conclusions, but without taking into account the results in cases 2 and 8. It is perhaps ironic that Head himself cites cases of his own which he could have advanced in support of Wernicke's localizations. Thus his case 14 was a case of syntactical aphasia, the lesion of which Head places "in and around the upper temporal gyri and the parts beneath them." He says of the case that "... his speech was jargon... his words poured out in phrases which... were in most cases incomprehensible. He could not repeat a sentence said to him.... He was unable to find names for common objects.... Comprehension of spoken words was obviously defective...." It is almost as if he were quoting directly

from Wernicke. Why Head so summarily dismissed Wernicke when his own evidence supported him must remain a mystery.

Wernicke closes his paper with some further comments. Baginsky, he notes, had spoken of 'centrifugal' and 'centripetal' forms of aphasia. But, he says, it is one thing to create hypothetical centers and quite another to construct a theory by converting known anatomical data into psychological form. He stresses the necessity of further anatomical knowledge. In the appendix he credits Hitzig with recently published views on aphasia similar to his own independently developed ideas.

At this point it is reasonable to consider why this paper had so great an influence. On might entertain various theories. A consideration of some features of Wernicke's career may help to clarify some of the possibilities. He was only 26 when the paper was written, without previous publications, unknown, without academic rank, of an undistinguished family, in a provincial university, without the support of major well-known figures. One can effectively dismiss the view that it was sheer prestige, academic authority or influence which were the sources of the effect of this paper. The growing prestige of German science in general would not account for the selection of his work over that of other better known German authors. A general tendency to accept diagram-making would again not be an adequate explanation since many writers of the day were producing their own diagrams.

Nor could the effect of the paper be attributed solely to Wernicke's later career. In the first place it had achieved a wide influence long before he himself had attained advancement. His academic progress was disappointingly slow and for several years in Berlin he was without academic connections. Having been appointed to the professorship in Breslau, he failed to move on from there to any of the more distinguished chairs. Hence, he never achieved great political influence within the German academic world. Furthermore he did very little further work on aphasia, leaving this area primarily to others. He advanced many other areas of neurology in later years but the aphasias and related disturbances were left to his students and others to prosecute. In fact, it is important to realize that he considered his major activity to be his psychiatric investigations eventually to be summarized in his text 'Grundriss der Psychiatrie in klinischen Vorlesungen'. But while his work on aphasia was so successful, his psychiatry was to have little influence, although his psychiatric writing

appeared during his period of greatest prestige. The same distinguished students who carried on his ideas in aphasia made the gesture of supporting their teacher's psychiatric teachings, but without enthusiasm. Yet Wernicke's psychiatry was also based on a simple diagram which failed of acceptance. The accusation of 'brain-mythology' was made about his psychiatric teaching, yet his anatomical ideas on aphasia succeeded despite the unsuccessful attempts to apply the same epithet to them. In fact, the most successful psychiatric investigator of the period was Emil Kraepelin, who differed fundamentally from Wernicke. His psychiatry was not based on anatomy, was without diagrams, and was thoroughgoingly based on clinical descriptions.

Since this was a period in which psychiatry and neurology were still intimately linked in Germany, the very same people who accepted Wernicke's diagrammatic anatomical approach to aphasia were simultaneously adopting a very different psychiatric teaching. This should make us wary of accepting any simple conclusion that there was a general tendency to accept anatomical explanations or simple diagrams without question. Furthermore, scientific controversy raged in the German academic life of that period and disagreements were aired freely and at great length.

Perhaps only a more thorough investigation will unearth the full reasons for the acceptance of Wernicke's first paper, and I can here only suggest some possibilities. In the first place his paper brought new clinical facts to attention. As I have already mentioned, Bastian and Schmidt were the first to call attention to the sensory varieties of aphasia (and Wernicke who had been unfamiliar with these works was later to give them full credit), but it was Wernicke's work which succeeded in disseminating knowledge of these forms. He stressed the existence of fluent aphasias in contrast to the previously well-known forms, and called attention to the error of diagnosing these as confusional or psychiatric syndromes. He brought all of these elements together into a syndrome described for the first time. Furthermore, he had resolved the problem of the existence of cases of aphasia without damage to Broca's area by showing that Meynert's demonstration of cases of aphasia with temporal lobe lesions was clarified by the existence of a syndrome of temporal aphasia different from that of frontal aphasia. He established the localization of his syndrome of sensory aphasia, to be confirmed by later authors. It is impor-

tant in viewing the paper to see it in the context of its period. Neuroanatomy was only beginning. The exact course of the auditory pathways was still unknown and Wernicke pointed out that the somesthetic pathways were still a mystery. Standards of clinical examination were hardly developed, and pathological examination was based on gross findings. This paper came at the beginnings but was to be one of the forerunners of the great revolution in neurology.

One final point is worth stressing in considering the response to this paper. Meynert had pioneered in the attempt to correlate the structure of the brain with its functions, and Wernicke attempted to carry this trend forward. Broca had first shown the importance of localized lesions in producing aphasia, and Meynert had probably been the first to bring some rationale to the localizations, but it was Wernicke's paper which made the first searching attempt to link the facts of anatomy with the facts of behavior in a way that permitted both prediction of syndromes and the organized testing of hypotheses. Like Meynert he gave the brain life. It was this trend that was continued in the work of many students both in Germany and elsewhere, and in good part must have accounted for the influential effects of this pioneering paper. The work of his own students such as Liepmann, and of others, such as Dejerine, who employing what was essentially the Wernicke model were able to apply it successfully to the analysis of other syndromes, must have continued to give viability to Wernicke's teachings.

Indeed we must remember that even such writers as Goldstein, apparently so different in theoretical outlook, did not, as I have pointed out elsewhere (Geschwind, 1964), wholly reject Wernicke's approach. Goldstein took over a major portion of Wernicke's teachings and his connectionistic approach, and indeed even employed this type of analysis in the approach to some problems. As I have said earlier he first learned about neurology from Wernicke. Over half a century later he wrote the following words of this early experience, "... his way of examining patients and his demonstrations were so elucidating and stimulating that we who had the good fortune to attend his clinics were deeply influenced in our further consideration of neurological and psychiatric problems. We could never forget him. His influence can be seen in the work of each of his many pupils, not a small number of whom became men of stature in their own right in the profession...." (Goldstein, 1953).

NOTES

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[1] Some recent authors, e.g., Penfield and Roberts (1959), have used the term to designate an area far larger than that referred to by Wernicke, but this is not in keeping with classical usage.

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CHAPTER XIII

SHRINKING RETROGRADE AMNESIA

PREFACE

Frank Benson and I decided to publish this case not because the phenomenon was new – Ritchie Russell and Peter Nathan had described it in 1946 – but because it had been neglected. Thus many animal experimenters had attempted to study the time course of consolidation of memory traces by such experiments as teaching the animal a task and after a variable interval using chemical or physiological methods to 'erase' the unconsolidated memory. The presumption in these experiments was that a failure of performance was equivalent to loss of the unconsolidated memory. This presumption failed, however, to take into account the possibility that such an experiment might merely reflect the production of a transient difficulty in retrieval. Only in recent years have animal experimenters come to be aware of this phenomenon and to study it.

SHRINKING RETROGRADE AMNESIA*

1967

ABSTRACT. A case of post-traumatic encephalopathy with a three month period of post-traumatic amnesia has been presented. Particular attention was given to the striking shrinking of retrograde amnesia which occurred after recovery of recent memory function had taken place and to the denial of illness seen in the course of the illness.

Two types of permanent retrograde amnesia are demonstrated by this case and other cases of memory loss, a long retrograde amnesia of several years' duration and a much shorter retrograde amnesia, usually of less than one day's duration. Retrograde amnesia of intermediate length appears to occur only during the period of shrinking of the retrograde amnesia. Long-term retrograde amnesia is associated with a continued defect in recent memory whereas short-term retrograde amnesia is present only in individuals with intact ability to learn new information. The fact that memories that are unavailable during long-term retrograde amnesia can return without a process of relearning shows that these memories are not lost, but that the ability to retrieve this material is lost during long-term retrograde amnesia. Thus it is suggested that the same anatomical substrate necessary for laying down new memories is probably necessary for retrieval of less overlearned or more recently acquired past memories. This has significant implications for animal experimentation dealing with memory, particularly those concerned with the time course of memory 'consolidation'.

Denial of illness was notable in this case up to the point that recent memory function returned at which time the denial abruptly ceased. This suggests that the denial was actually a confabulation caused by inability to learn new information, specifically the details of the present illness. After recovery of memory function we were able to reproduce the denial of illness, the recent memory defect and lengthening of the retrograde amnesia by use of intravenous amytal, again suggesting the linkage of these disturbances.

Memory disturbance has long been recognized as a key feature of post-traumatic encephalopathy but one feature of this abnormality, the shrinking of retrograde amnesia, has received scant attention. First described by Russell (1935), and in greater detail by Russell and Nathan (1946), this phenomenon has been neglected by both clinicians and animal experimenters. Recently Deutsch *et al.* (1966) have demonstrated a similar phenomenon in animal studies. We feel that recognition of shrinking retrograde amnesia is of distinct importance in the clinical appraisal of post-traumatic memory disturbance and in addition has several significant implications for any theory of memory function. We present here a case of post-traumatic memory loss which clearly demonstrates shrinking of

retrograde amnesia and discuss both the clinical and theoretical considerations of this phenomenon. Another clinical feature commonly seen in acute traumatic encephalopathy, denial of illness, was also present in this case and will be discussed.

I. CASE REPORT

J.R., a 33-year-old right-handed man, was admitted to the University Hospital, Boston, Massachusetts, on 26 November 1965 with a large subcutaneous haematoma in the right temporal-parietal region and deep lacerations across the forehead. He was stuporous but not totally unconscious and there was a mild right hemiparesis. No history was available concerning the cause of injury.

Later additional history was made available. The patient was married and had a family living in Washington, D.C., but two years earlier he had separated from his wife and moved to Boston. He had worked as a bus driver in Washington. Since his arrival in Boston the patient had held two jobs, first as a messenger for a drugstore and later as a labourer in a mattress factory. He had finished work on the evening of 25 November and was to have taken a bus trip to Washington. He had not taken the bus and it was assumed, although never definitely proven, that he had gone on a binge. The patient's drinking history was noteworthy; he was not a regular drinker, but on rare occasions he would go on binges lasting several days. He had never needed treatment for alcoholism or its complications. In fact his health had been excellent, his only medical complaints for many years having been minor disturbances of the feet and vision.

During the first hospital week the state of consciousness varied from stupor to semicoma, the right hemiparesis persisted and it was felt that he was aphasic. Radiographs of the skull showed no fracture, lumbar puncture had normal pressure, no cells, and protein of 68 mg per 100 ml. An electroencephalogram was abnormal and was considered compatible with a subdural haematoma. A brain scan and a left carotid arteriogram were performed and both were considered to be normal. After one week the patient began to improve, the state of consciousness lightened, the paresis receded and then disappeared but a consistent language disturbance remained. Originally described as pure jargon, the speech later was

described as fluent with paraphasia. In addition there was an abnormal behaviour pattern with jocularity, facetiousness, unreliability, and topographical disorientation in the hospital.

He was seen in consultation by members of the Boston University Aphasia Research Unit on 26 December 1965, exactly one month after admission. By that time he was totally free of disturbance of primary motor or sensory functions and there was no evidence of visual field abnormality. He was bright, alert and apparently attentive to the examination but notably distractible. Digit span was recorded at 6 forward. When questions were asked he would respond, not with answers, but with additional questions as to why the examiners were seeking such information. He was totally disoriented for time, giving dates at least three or four years before the time of the examination. When asked to give the date the patient would often evade the question suggesting that the examiners actually knew the date and that he did not need to provide this information. He consistently stated that he was in Washington, D.C., despite being told regularly that he was in Boston. If asked about employment he would reply that he was working as a bus driver in Washington. He was totally unable to remember the names of the physicians and nurses attending him. He could not retain three unrelated words for three minutes. When questioned as to the type of building that he was in he would deny that it was a hospital giving a variety of responses some of which were paraphasic. Thus one reply was "some kind of malingua place – you use it a lot in South America". If the examiner insisted that the building was a hospital the patient would not argue. If he was immediately asked why he was in the hospital he would either claim that he was visiting or would present a seemingly irrelevant disorder as the cause of hospitalization. On several occasions he stated that he was in hospital because of eye problems; at other times he mentioned difficulties with his feet or legs. He adamantly denied any difficulties in the use of language or memory when specifically questioned concerning these.

His speech was fluent and well articulated but empty of meaning and both literal and verbal paraphasias were present. Comprehension of spoken language was considered normal for ordinary conversation. Repetition and serial speech were normal. He could name simple objects on confrontation but had difficulty if asked to name parts of objects. Thus the crystal on his watch became 'clystal', the watch stem was called a

'spicket', and the lead of a pencil became 'rubber'. He was able to give the names of only three out of 10 colours. He had great difficulty in presenting word lists; thus he could list the names of only two makes of automobiles in 60 seconds and named only 'ocelot' and 'bear' when asked for a list of animal names. Reading (aloud and for comprehension) was adequate. Orthography was good but the content of his writing was circumlocutory and empty. Right-left orientation and finger localization were intact. He had great difficulty in construction tests, particularly when asked to copy drawings. He was able to calculate.

The patient was transferred to the Boston Veterans Administration Hospital on 27 December 1965 where it was immediately apparent that memory disturbance was the most severe disability. He wandered off the ward, could not find his own bed, would lie down in any bed that was handy, and was frequently seen looking for objects in other patients' bedside stands. At one time he was wandering in the hospital parking lot, at another time he was watching television three floors below his own ward. Because of this tendency to wander he was transferred to a locked ward on 29 December 1965. During his stay on the locked ward, the jocular, facetious, evasive behaviour, the inability to remember the names of the physicians, the nurses or the hospital, and complete disorientation for time persisted. During this period there was improvement in language so that his speech became more definite and the previously noted paraphasia disappeared. He consistently stated that he was in Washington, D.C., that his last job was that of a bus driver, and he denied illness or memory defect.

An electroencephalogram done during this period was mildly abnormal with suggestion of a slow wave focus in the left posterior temporal region. A brain scan on 20 January 1966 was normal. A pneumoencephalogram on 17 January 1966 showed the size of the lateral ventricles at the upper limits of normal, the fourth ventricle slightly enlarged but the third ventricle of normal size. The left temporal horn was slightly larger than the right but it was not felt that the pneumoencephalogram showed evidence of either mass lesion or hydrocephalus. Spinal fluid taken at that time showed normal pressure, total protein of 48 mg per 100 ml., and no cells were present.

The patient's status remained essentially the same except for the improvement in the use of language until about the first of March 1966. During a period of one or two days the ward nurses noted a marked

change in his behaviour, for the first time caring about the way he dressed and handled his food, asking them for their names and then correctly using their names later. Re-evaluation was performed on 2 March 1966, at which time he was able to recall three unrelated words after three minutes, to remember the name of the doctor, to remember the name of the hospital after it had been given to him, to recite the names of the ward nurses, and to tell the number and location of the ward. He still manifested a retrograde amnesia since he could not remember moving away from Washington, D.C., but was willing to accept that he was now a patient in a Boston hospital. He no longer denied head injury but stated that he had only recently been told of this injury. He was transferred back to the Neurology Service and had no difficulty now in locating his bed, in getting about the ward and the entire hospital. His behaviour pattern also changed as he became less evasive but remained jocular and facetious. Language evaluation at this time was normal except for minimal evidence of word finding difficulty. He was able to give the names of eight animals in a period of 60 seconds, only slightly below normal levels.

During the remaining period in hospital, the retrograde amnesia consistently improved. Thus when he first returned to the Neurology Service he was unable to remember living in the Boston area. Three or four days later he spontaneously recalled separating from his wife and moving to Boston. Several days later he remembered the job that he held when he arrived in Boston and the name of his employer. He was unable to remember any other job in Boston. Within a few days, however, he recalled the second and last job but was still vague as to how long he had worked there. Before discharge the retrograde amnesia had cleared to the point that he remembered quitting work on the day before the injury, approximately 24 hours before admission to the Unversity Hospital. It is likely that the patient began drinking at that time but this was not confirmed by the patient nor any other history available to us.

On the day before discharge an amytal interview was performed. The patient was given approximately 400 mg of sodium amytal intravenously, sufficient to produce both drowsiness and nystagmus. He was questioned about orientation and retested for aphasia. There was a recurrence of the tendency to locate the hospital in Washington, D.C., and to deny that he had suffered a head injury. He again stated that the had been brought

to the hospital by his mother because of eye problems. The aphasic deficits noted when he first came to this hospital did not reappear under the influence of amytal. The interview was initiated by two examiners who later were joined by a third person. The day following the interview the patient claimed no memory of the interview and did not remember the third person joining the group despite the fact that he had conversed freely with this examiner during the interview. At the time of discharge the patient still had total amnesia for a period starting about 24 hours before admission to hospital at the University Hospital until he suddenly found himself in the locked ward at the Veterans Administration Hospital. He was quite aware of details from that point on. Following discharge the patient returned to Washington, D.C., where he was seen on three occasions in a Veterans Administration clinic. There was no evidence of aphasia or memory deficit so he was discharged from the clinic.

II. COMMENT

This case demonstrates several clinical features which deserve discussion. The most dramatic observation was the rather rapid reappearance of the ability to learn new material followed by a slower but progressive shortening in the duration of the retrograde amnesia. This course in the recovery from traumatic memory loss (shrinking retrograde amnesia) has been described previously (Russell and Nathan, 1946) but is not widely recognized in the literature of memory defects. This phenomenon has important implications for both the clinical and experimental evaluation of memory loss.

Changes in memory following head trauma are most often described as consisting of two types of 'amnesia', post-traumatic (anterograde) and retrograde. The first of these, post-traumatic amnesia, is defined as 'the interval during which current events have not been stored' (Russell and Smith, 1961). To the clinician post-traumatic amnesia is most vividly demonstrated when a patient, evaluated long after recovery from head injury, describes total amnesia not only for the period of unconsciousness but for many additional days or weeks, a period during which the patient had apparently regained normal mental function. Older authors described 'recovery of senses' (Jackson, 1932) or of 'vigilance' (Head, 1926) indicating that this was the termination of impaired consciousness but it

must be emphasized that a patient may be entirely alert and the 'impairment of consciousness' in post-traumatic amnesia may be confined to an inability to store current events.

The second major division of post-traumatic memory loss is a retrograde amnesia, an inability to recall events preceding the injury. After full recovery the retrograde amnesia is usually quite short, often encompassing only a few seconds before the injury and only rarely exceeding a few minutes. In our patient the permanent retrograde amnesia could not be ascertained since we did not know the exact time of injury, but it could not have exceeded 24 hours. Our estimation of the retrograde amnesia is further complicated because our patient may well have been intoxicated for many hours preceding the trauma. In patients who suffer a permanent post-traumatic loss of the ability to acquire new memory, however, a much different retrograde amnesia is noted, usually covering several years befor the injury. In this latter characteristic the post-traumatic retrograde amnesia is similar to that seen in other disorders with permanent memory loss such as the metabolic Korsakoff's syndrome in which one usually finds a retrograde memory loss of several years before the onset of recognized memory disturbance. An interesting dichotomy is thus present in retrograde amnesia, one type with an amnesic duration of seconds or minutes, and the other with retrograde amnesia lasting at least several years. The patient with shrinking retrograde amnesia thus represents a transitional form between those with long and those with short retrograde amnesia. The observation that permanent retrograde amnesia is usually either very long or very short and that a permanent retrograde amnesia of, let us say a month's duration, is extremely unusual, is of great interest in the theory of memory function and will be discussed in detail.

As post-traumatic amnesia and retrograde amnesia are terms usually used in describing traumatic memory loss another set of terms is indicated for theoretical discussion. The three categories of immediate recall, recent memory, and remote memory have been recognized for many years and have been proved to indicate separate and distinct functions.

Immediate recall (also called immediate memory and short-term memory) has been studied intensively (Brown, 1964). It is described as the ability to retain material for 10 to 30 seconds and is differentiated from the retention of material for several minutes or longer. This type of 'memory' does not entail a permanent coding of the material.

Recent memory can be defined as the ability to retain new material for several minutes or longer. Stated another way, recent memory is the ability to 'learn' new material. It is generally accepted that pathological changes in the limbic system (hippocampal region and mammillary bodies are the most frequently cited) are present in cases with loss of recent memory (Barbizet, 1963). It has also been suggested that these structures must be intact for the consolidation of newly learned material.

Remote memory may be considered to be the ability to recall material learned before the onset of the patient's illness. Included in this previously learned information are basic capabilities such as use of language and numbers, feeding and dressing skills, and significant past history (i.e., birth and marriage dates, career milestones etc.) plus purely learned material (i.e., names of presidents, important historical dates, etc.).

A period of post-traumatic amnesia clearly corresponds to a period in which there is loss of recent memory function. Such a loss may be limited in time or may be permanent. A corresponding total loss of remote memory function does not occur. Even in the most severely involved patient with a Korsakoff's syndrome much of remote memory is retained. In general the loss for the years immediately preceding the illness is most severe. Remote memory therefore is not involved in as global a fashion as recent memory. It would appear that older, probably overlearned material (e.g., language, the activities of eating, dressing, etc.) is more resistant than more recently acquired material. The inability to recall material acquired in the period preceding the onset of the illness is usually ascribed to the fact that the memory of this material is not 'consolidated' and therefore is more readily wiped out by, for example, a head injury. This explanation carries with it the implication that the process of 'consolidation' may take several years.

It appears to us that the thesis of the abolition of poorly consolidated memory traces as the explanation for retrograde amnesia does not explain the findings of shrinking retrograde amnesia. It is obvious that many of the remote memories which the patient is incapable of recalling in the period immediately following his head injury are not abolished, since the patient does recall them at a later date. The disturbance is therefore a failure of retrieval rather than a loss of established memories. By contrast the post-traumatic amnesia seems most likely to represent not a failure of retrieval but a failure to establish new memory traces. The permanent

post-traumatic retrograde amnesia of seconds' to minutes' duration does, however, probably represent the true abolition of memories not yet consolidated.

According to this view remote memory would fall into three groups. There is a period of seconds to minutes in which a memory is fragile and may be permanently abolished. There is a longer period in which the memory is consolidated but in which retrieval may be impaired. Finally many memories, especially old and overlearned ones, may be retrieved despite influences which abolish the most recently acquired memories and affect the retrieval of many later memories.

It seems possible, furthermore, that the retrieval process depends on the same system that is necessary for the laying down of new memories. This would explain the fact that the shrinking of retrograde amnesia runs generally parallel to the recovery of recent memory function. It would also account for the fact that a patient with a permanent recent memory deficit has a permanent long retrograde amnesia. Furthermore, the fact that in metabolic Korsakoff's syndromes a permanently long retrograde amnesia is associated with a permanent recent memory loss militates against the assumption that head injury has some unique effect on memory. It would therefore seem probable that those limbic structures which are necessary for the laying down of new memories are also necessary for the retrieval of the more recent or less over-learned older memories.

The permanent long retrograde amnesia of either the metabolic or post-traumatic Korsakoff's syndrome is then a retrieval deficit. It is thus conceivable that by some means it might be possible to make these memories capable of retrieval although this would not help the patient materially since he would still be suffering from inability to lay down new memories. The consideration must be entertained, however, that in other disturbances of the higher functions, e.g., some aphasic syndromes, retrieval deficits are present, reversal of which, by as yet unknown means, might markedly improve the patient's functional capacities.

Retrograde amnesia and the dramatic phenomenon of shrinkage of retrograde amnesia have important implications for animal experimentation dealing with memory. Until recently the spontaneous return of memory after a lapse of time following artificially induced amnesia had never been recorded in animals (Deutsch, 1962). A series of experiments reported by Deutsch *et al.* (1966) suggested a 'memory loss' which disap-

peared with time, a finding which agrees with the clinical observations recorded in the present case. This shrinkage of retrograde amnesia was not recognized in many of the earlier animal experiments and theories of memory function based on such incomplete experiments must be viewed with some doubt. Particular question must be raised concerning those studies which purported to find that the process of 'consolidation' was prolonged. It seems likely that most of these studies actually demonstrated difficulties in retrieval.

The late recovery of memory function is of significance clinically. The behaviour problems associated with memory loss demand protective care of the type most often provided by psychiatric institutions. Admission is usually by commitment and recovery of memory function is unexpected, leading to prolonged incarceration. Thus the potentiality of excellent recovery of memory function, even many months after brain injury, deserves emphasis.

This case demonstrated another dramatic phenomenon, denial of illness, which has been recognized and discussed for many years. It has been suggested by some that a parietal lesion was necessary for such denial (Schilder, 1934; Nielsen, 1938), by others that denial was a manifestation of clouding of consciousness (Spillane, 1942; Sandifer, 1946), or that denial of major neurological defect suggested impaired recognition of body scheme (Gerstmann, 1942; Critchley, 1953). Weinstein and Kahn (1955) suggest that denial is actually motivated and represents a psychological defence mechanism occurring in a patient with widespread dysfunction of the nervous system who is unwilling to accept the presence of a major deficit.

The present patient had clear denial of illness when first seen and the denial persisted, essentially without change until the post-traumatic amnesia cleared. During this time the patient adamantly refused to admit head injury, language disorder, or memory loss, and if pushed by interrogation would state that he was in the hospital because of visual difficulty or foot trouble. There was consistent disorientation for time, most often evasive, but if firmly pushed the patient would give a date several years before the time of his injury. He consistently insisted that he was in Washington, D.C., his former home.

Taken out of context these findings could be considered motivated with the patient actively denying his obvious defects. It seems preferable, how-

ever, to describe this as confabulation, an attempt by the patient to fill a void in his knowledge of present occurrences with past knowledge. This fits a current theoretical view (see, for example, Barbizet, 1963) which suggests that confabulation is an attempt on the part of a patient, unable to learn new information, to provide requested information from the only source available to him, his past memory. Our patient continued to deny his neurological defects and remained disoriented until he rather rapidly regained normal recent memory function at which time all denial symptoms disappeared. In our opinion the denial and memory loss were probably results of the same process. Thus it is not entirely correct to state that our patient denied his illness but would be more accurate to say that he was unable to remember the present difficulty and attempted to fill this void by providing older, previously overlearned information.

A defect in word finding was clearly demonstrated in this patient and was such a prominent feature in the early part of the hospital course that at least one competent examiner considered the major difficulty to be anomic aphasia. Weinstein and Kahn noted word finding defects in many of their patients who denied illness. In their opinion, the naming defect appeared to be more pronounced for hospital oriented items (i.e., thermometer, stethoscope, urinal, etc.) than for other objects. They suggested that this was not a true naming defect and that the 'paraphasia' demonstrated in these patients was truly a non-aphasic misnaming of stress-producing items. In the present case this theory cannot be substantiated. It is true that our patient had difficulty in naming hospital oriented items but he had just as much difficulty naming other items. While it would be tempting to link his memory disturbance with his anomia it is difficult to do so since our experience as well as that of others confirms the existence of severe memory loss in adults without anomic disorders. It is most likely that our patient's anomic disturbances were secondary to cerebral contusion.

Finally it is worth discussing the results demonstrated by the amytal interview. This technique was used by Russell and Nathan and was also used extensively by Weinstein and Kahn. In the present case we were interested mainly in the possibility of reappearance of aphasic symptoms so we did not attempt to demonstrate retrograde amnesia nor did we realize during the interview that we had actually produced a period of recent memory loss. However, in an interview with the patient the follow-

ing day it was quite apparent that he had no memory whatsoever of occurrences during the interview and in fact did not realize that a third person had joined the interview team, a person whom he knew very well, and who had talked to him during the interview. Quite possibly better observation on our part would have demonstrated a distinct reappearance of retrograde amnesia but even our limited observations suggested its recurrence. One of the most significant parts of the interview was the reappearance of the denial of illness. The patient strongly and adamantly denied that he was in the hospital because of any head or brain injury and again told the story of being brought to the hospital by his mother because of eye problems. Before the amytal interview the patient was no longer telling this story and following the amytal interview he was quite aware of the reason for hospitalization. Thus with amytal we were able to produce many of the findings that were seen during the acute state of the disorder and suggest that one of the effects of amytal is to impair the function of those regions involved in new learning and in the retrieval of more recent (less overlearned) past memories.

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NOTES

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CHAPTER XIV

THE APRAXIAS

PREFACE

The apraxias have a special importance in the history of callosal syndromes, since it was the study of these disorders which led Liepmann to his remarkable analyses of syndromes of disconnection. The apraxias represent today that group of disorders of the higher functions which are best understood anatomically. The paper reprinted here represents a fairly elementary summary of the material in 'Disconnexion syndromes in animals and man'. Since the publication of this paper I have had the opportunity to develop my ideas much more extensively. A much more complete discussion will appear in a future publication.

THE APRAXIAS*

1967

The apraxias are disturbances which are characterized by "the incapacity for purposive movement of the limbs despite retained mobility." This definition is that of Liepmann who first used the term apraxia in its modern sense (Liepmann, 1900, 1906). It excludes from consideration all forms of weakness, disturbances of tone or posture, reflex abnormalities whether elementary or complex (e.g., the movement disorders resulting from the grasp reflex), disturbances of coordination or abnormal movements, as well as the failures of movement which result from complete deafferentiation of the limb (which presumably breaks the linkage of γ and α motor neurons, thus essentially paralyzing the usual pathway for innervation of the α neurons). Some authors have attempted to deny the existence of apraxias satisfying these restrictions but it is clear, both from the literature and from some of the examples to be cited in this paper, that such cases exist. In order to show that a failure of movement is apraxic we must show that a movement not made under one set of conditions can be performed in other conditions, or that movements of equal or greater complexity can be made under other circumstances.

We must also show that the failure to make a movement is not the result of incomprehension of a command, the result of unwillingness of the patient to cooperate with the examiner, or non-recognition of objects shown to him. In the discussion of specific apraxic syndromes later on in this paper it will be shown how all the requirements specified here may be met.

Liepmann provided the initial analysis of the apraxias on an anatomical basis. My own investigations have convinced me of the fundamental correctness of his views. I will attempt here to present a model which is essentially the one advanced by Liepmann with certain rather important modifications and additions. A much more detailed discussion of this problem will appear elsewhere (Geschwind, 1965).

The opening paragraphs of this paper have suggested that the apraxias are not complex motor or complex perceptual disturbances, nor are

they the result of incomprehension or uncooperativeness. In order to understand the apraxias we must see them predominantly as *associative* disturbances, that is to say, as failures of connection between higher regions of the nervous system, in particular between different parts of the cerebral cortex. It is therefore necessary first to consider the pattern of connections between cortical regions.

Flechsig (1901) pointed out that the primary cortical motor and sensory regions, i.e., those which receive or send long subcortical projections, have no direct connections one to the other. Each of these elementary regions has cortical connections only with immediately adjacent cortical regions, the so-called association areas. The association areas of the brain are the only cortical regions that have iong connections with other parts of the cortex. Let us consider in the light of this principle the simple problem of how one might carry out a learned motor act to a learned auditory stimulus.[1] The auditory stimulus will travel along the auditory pathways and will eventually reach the primary auditory cortex (Heschl's gyrus) on the superior surface of the temporal lobe. From here it is relayed to the auditory association cortex which lies adjacent to Heschl's gyrus. That part of the auditory association cortex which lies in the posterior portion of the first temporal gyrus on the left side is the classic Wernicke's area.

From the auditory association cortex there are probably either no direct connections to the primary motor cortex or only a small number of such connections. There are, however, connections which probably arise in the auditory association cortex and run to the cortex lying anterior to the classical motor cortex (Sugar, French and Chusid, 1948) and which we may call 'motor association cortex'. The pathway between the auditory and motor association cortexes is part of the classical arcuate fasciculus. From the motor association cortex there are then connections to the classical motor cortex. Figure 1 shows the pathways mentioned.

We can now consider the specific problem of how one carries out a verbal command with the *right* hand. The pathway used lies in the left hemisphere and in fact is the one we have just specified, running from Wernicke's area to the motor association cortex of the left side and finally to the left motor cortex. How do we perform the apparently similar task of carrying out verbal commands with the *left* hand? Since in the great majority of people the comprehension of language is depen-

dent on Wernicke's area (lying in the left hemisphere), the command must first reach Wernicke's area and be relayed from there. The problem now is to trace a pathway from Wernicke's area, which lies on the left side, to the right motor cortex. Most evidence indicates that fibers in the corpus callosum connect roughly symmetrical areas in the two hemispheres, and that pathways running obliquely in the callosum are rare. This requirement that connections between the right and left hemispheres be between symmetrical regions puts certain limitations on the pathway from Wernicke's area to the right motor cortex. When this

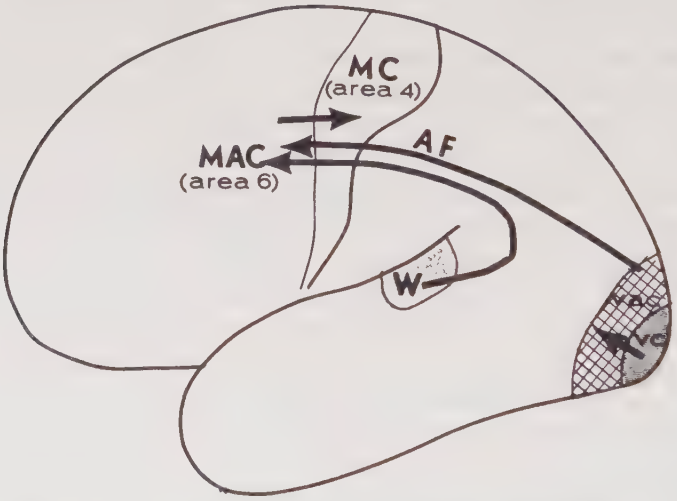

Fig. 1. Lateral view of left side of brain. AF = arcuate fasciculus, MAC = motor association cortex, MC = motor cortex, VAC = visual association cortex, VC = visual cortex, W = Wernicke's area. The arrows indicate major connections of the areas shown.

limitation is considered, it is apparent that there are two possible pathways, and these are shown in Figure 2. It seems likely, however, that the pathway marked *a-b-c* is not used. The diagram shows this path running from Wernicke's area to the symmetrical region on the right; from here it runs to the right motor association cortex and finally to the right motor cortex. Evidence from both anatomical (Krieg, 1963) and physiological studies (Curtis, 1940; Bonin and Bailey 1947; Bailey *et al.* 1950) indicates that in the monkey and chimpanzee this part of the auditory

association cortex has few or no callosal connections. It is not unlikely that the same holds true in man. It therefore seems likely that the pathway marked *1-3-c* in Figure 2 is the one actually followed. This pathway runs from Wernicke's area to the left motor association cortex, then via the corpus callosum to the right motor association cortex, and from there to the right motor cortex, and therefore involves at least three neurons. The diagram of Figure 2 is very simple, yet it serves to explain a major portion of the apraxic disturbances seen by neurologists. We can recount these by simply running through the diagram.

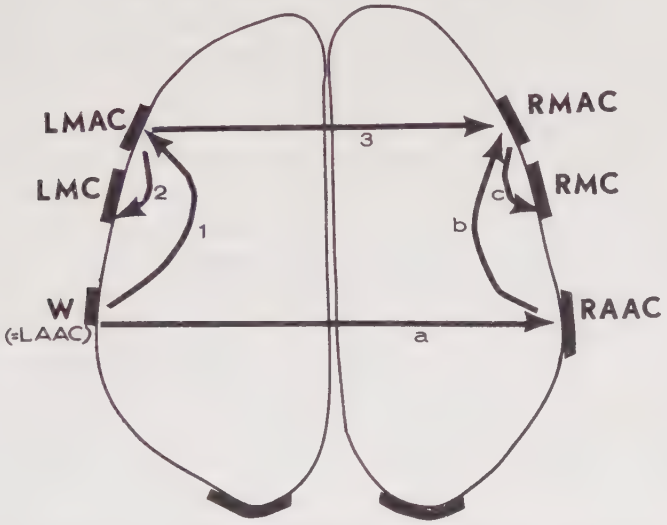

Fig. 2. Brain in horizontal section viewed from above. LMAC and RMAC = left and right motor association cortex, LMC and RMC = left and right motor cortex, LAAC and RAAC = left and right auditory association cortex, W = Wernicke's area. The arrows indicate fiber pathways.

Consider a lesion at *1*, i.e., involving the left arcuate fasciculus. The patient with this lesion will fail to carry out correctly motor commands with either his right or left limbs. It is obvious, however, that he will have no elementary motor or sensory disturbance. Furthermore, although his failure to perform may at first suggest to the examiner that he does not comprehend, he will manifest normal comprehension in several ways: (1) He will select the correct movements from a series of move-

ments made by the examiner and reject the incorrect ones. (2) He will show comprehension of very complex verbal material (which does not involve the carrying out of a command) by giving appropriate verbal responses.[2]

Consider now a lesion involving the left motor association cortex (LMAC in Figure 2). This destroys the callosal pathway from the left to the right motor association cortex. Generally we see this lesion in patients with damage involving Broca's area and the left motor cortex. The patient usually demonstrates a right hemiplegia and a Broca's aphasia. He fails to carry out verbal commands with his intact left hand and is therefore frequently considered incorrectly to show a comprehension deficit. If the patient is tested by the methods suggested above, comprehension deficit can be ruled out. This type of apraxia, that of the left hand in the patient with a right hemiplegia and a Broca's aphasia, received the name of 'sympathetic dyspraxia' and is much the most common variety of apraxia of the limbs, being present in a sizable percentage of patients with Broca's aphasia. It was first described by Liepmann (1905) and attention has recently been called to its frequency (Geschwind, 1963).

The two forms of apraxia I have just discussed are, for reasons already mentioned, usually but not invariably accompanied by aphasic speech disturbances. With a lesion at *3* (in Figure 2) we get a much more isolated disturbance. This is a lesion of the corpus callosum itself rather than one of its cells of origin. This too was first described by Liepmann (Liepmann and Maas, 1907). Mrs. Edith Kaplan and I (Geschwind and Kaplan, 1962) have had the opportunity to study such a patient extensively. This patient wrote correctly with his right hand but aphasically with his left hand. He carried out verbal commands correctly with the right hand but failed with his left hand. The possibility that this was any form of general comprehension disturbance or uncooperativeness was ruled out by his carrying out verbal commands correctly with the right hand. The possibility of any elementary motor or sensory disturbance in the left hand as the cause of these phenomena was ruled out by the fact that he would imitate the correct movements perfectly if they were demonstrated to him by the examiner. This case is a very clear example of a disturbance occurring as the result of disconnection of the speech area from the right motor area.

Having given this general anatomical overview, let me cite one or two

special problems. Of all apraxias *facial apraxia* is the most common, being present in most cases of Broca's aphasia. It occurs in other situations as well. Thus in the forms of apraxia involving lesions deep to the left supramarginal gyrus or the left motor association cortex, there is nearly always apraxia of the face. It may be seen in isolation, i.e., without significant limb apraxia, particularly in cases of Broca's aphasia. It represents only a special form of sympathetic dyspraxia in which those parts of the pathway involving the limbs are relatively spared but not those involving the face, which, lying lower down in the hemisphere and therefore nearer to Broca's area, are more likely to be affected in a Broca's area lesion.

In some patients with lesions in any of the sites indicated above, complex *whole body movements* may be performed to command while limb and face movements fail. While we cannot discuss this problem in detail, there is evidence which suggests that whole body movements may be mediated by a pathway which runs directly via the so-called bundle of Türck from Wernicke's area to a group of pontine nuclei which then project to the vermis of the cerebellum. This pathway will of course be spared by the lesions we have so far considered. Further study will be needed to demonstrate whether this is indeed the correct mechanism for the sparing of whole body movements.

I have concentrated in my earlier discussion on failures to carry out movements to verbal command. When we examine patients with this type of disturbance, we find that generally they show the following pattern. They perform most poorly in carrying out movements to verbal command. Generally there also do poorly in carrying out movements in imitation of those made by the examiner although sometimes this type of movement is preserved. Finally, there is usually much better performance when the movement is made in the presence of the usual stimuli to its production (e.g., instead of asking the patient to show how a hammer is used, the examiner actually hands him a hammer; instead of asking him to pretend to blow out a match, he is presented with a lighted match). There are, however, a small group of patients who do poorly even in this situation. I have not personally seen patients who fail in the presence of the actual object but succeed in carrying out the task on imitation or to verbal command. Nor have I seen a patient who failed to imitate but succeeded in carrying out the task to verbal command. I have,

however, commonly seen patients who fail on verbal command but perform normally in the other two situations. Most common is the patient who does poorly on verbal command and imitation but does better in the presence of the normal stimuli to the act.

How do we explain this pattern? We cannot fully answer this question but can only speculate on some aspects of it. The first point to consider is that carrying out a task under visual control (e.g., imitating a movement made by the examiner) probably involves a pathway running from visual association cortex to motor association cortex and therefore makes use of fibers which also run in the arcuate fasciculus. These are shown in Figure 1. Hence a lesion of the arcuate fasciculus will involve these as well as the auditory-motor fibers. We would then have to assume that the left hemisphere was dominant for these visual-motor connections (just as it is for the verbal-motor connections mentioned earlier) on the basis of the fact that most patients with a left arcuate fasciculus lesion develop a *bilateral* difficulty in imitating the examiner's movements. This indicates that the visual-motor connections of the right hemisphere cannot generally be used for this purpose and that the visual-motor pathway must go by way of the left motor association cortex, as is true of the auditory-motor pathway. This view is further corroborated by the fact that some cases of callosal disconnection fail to perform with the left hand not only to verbal command but also on imitation of the examiner. It appears not unlikely, however, that there are some people who can in fact make use of the visual-motor connections on the right side. Thus some people with callosal disconnections who fail to carry out verbal commands with the left hand do very well on imitation, e.g., the above-cited case of Geschwind and Kaplan (1962). The available evidence does not suggest that this is a difference in the intensity of lesion, but is rather a matter of individual variation. Since we know that there is a minority of people for whom dominance for speech is not so powerfully localized in one hemisphere, the same is probably true for this type of visuomotor dominance. For further details on the problem of the apraxias the reader is referred to the more complete discussion already cited (Geschwind, 1965).

PHILOSOPHICAL IMPLICATIONS

Because the interest of this symposium is concentrated to a great exten

on philosophical problems, permit me briefly to dwell on some implications of my discussion which perhaps may be of some philosophical interest.

Consider for a moment the patient of Geschwind and Kaplan cited earlier who failed to carry out verbal commands with the left limbs. Many observers who saw this patient were perplexed. They argued that if the patient both understood the command and could move his left arm, he should have been able to carry out the command. The source of the difficulty is in the use of the word 'patient', which is in fact used ambiguously in the preceding sentence. Thus it is perhaps correct in a certain sense to say that 'the patient understood' and 'the patient could move his left arm', but at another level it is clear that the term 'the patient' is ambiguous. If we point out more correctly (albeit clumsily) that 'the patient's left hemisphere understood' but that it was 'the patient's right hemisphere which could move his left arm', the problem disappears. The patient is thus not a unity. One could perhaps ask whether the patient's consciousness was multiple or single. I would find it difficult to accept that this patient had a unitary consciousness. This is not, however, to deny that in the normal intact person consciousness may be unitary. It appears, however, that this unitary quality may not hold under certain conditions of disease such as those I have outlined.

Similarly, these cases should give us pause in accepting readily a point of view which had a powerful influence in all the behavioral sciences, particularly in the period between the wars. Workers in these areas were enjoined to look at the patient as a whole man and not to view him as compartmentalized. I would not question the value of looking at the whole man under certain conditions, but it is clear that there are circumstances in which this approach cannot only be inappropriate but actually actively misleading to the investigator. To look at the patient as fragmented may be extremely fruitful under certain conditions of disease. Indeed, perhaps even the normal intact person is not always so much of a whole as we have been led to believe. This may be particularly true in early childhood before associative connections have been fully established.

A final and related point concerns the value of introspection. When we asked our patient why he failed to carry out certain commands with his left hand, we got answers such as, "I never used my left hand," "I never

learned to write with my left hand," or "I'm right-handed," all of course inadequate as explanations for the reason that a normal intact right-hander readily performs commands and writes (clumsily but correctly) with his left hand. Further reflection reveals, however, that it was probably useless to ask the patient to 'introspect' about what was going on in his right hemisphere. We were asking for verbal replies and therefore demanding a response from the speech area which was in his left hemisphere. But since the right hemisphere was disconnected from the speech area, there was no way that the speech area could recount to us what was going on in the right hemisphere. This means that the value of 'introspection' may be seriously limited in many situations in which there are lesions in the nervous system. Furthermore, if we ask the patient for introspections in such an instance, he is quite likely to give us confabulatory responses. Investigators of the higher functions of the nervous system have in fact been misled at times by such responses. It is important to have an active realization of the impossibility of getting an adequate verbal account of the activities of a part of the nervous system which is disconnected from the speech area. This is not to deny that in the normal intact man introspection may be a most useful technique.

NOTES

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[1] It is worth stressing that I am speaking here of *learned* responses. Certain motor responses to auditory stimulation, e.g., sudden head-turning to a loud sound, are probably unlearned and very probably do not involve the cortical connections being discussed here. Some learned motor responses may also use direct pathways from cortical to subcortical regions but such movements are probably limited in delicacy and complexity.

[2] Most of the patients with lesions in the arcuate fasciculus show aphasic disturbances in speech because the lesion almost invariably cuts the connections from Wernicke's to Broca's area: this is the classical conduction aphasia. These patients, however, show excellent comprehension which can be demonstrated in many ways, e.g., by having the patient indicate with a simple 'yes' or 'no' (an ability usually preserved) the answers to complex questions.

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CHAPTER XV

DICHOTIC LISTENING IN MAN AFTER SECTION OF NEOCORTICAL COMMISSURES

PREFACE

Doreen Kimura's modification of the dichotic listening technique of Broadbent for use as a means of studying the functions of the individual hemispheres represents a major contribution to the study of the human brain, since it is completely harmless and can be used in normals as well as patients. Mr. Robert Sparks and I made the first study of dichotic listening in a patient with a callosal section, whom we were able to study through the kindness of Dr. Joseph Bogen, Dr. Roger Sperry and Dr. Michael Gazzaniga. Our study was based on only one patient, and we were therefore pleased that a subsequent study of a larger series of callosally sectioned patients by Milner *et al.* (1968) confirmed our findings.

The disorder of dichotic listening in our patient highlighted for us a point that had probably not received adequate attention in earlier studies, i.e., the importance of the corpus callosum in this task. It also enabled us to explain the apparently paradoxical finding of suppression of the *left* ear in dichotic listening tasks in many aphasics (suffering almost always, of course, from left hemisphere lesions) which we discuss briefly in this paper but which was presented in greater detail by Sparks *et al.* (1970).

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DICHOTIC LISTENING IN MAN AFTER SECTION OF NEOCORTICAL COMMISSURES*

1968

ABSTRACT. Studies are presented of verbal dichotic listening and summation tasks in a patient who had undergone complete section of the neocortical commissures for epilepsy. The patient showed complete extinction of signals received by the left ear, this being greater than that evidenced by any of fourteen patients with right hemisphere lesions. It is suggested, both on the basis of earlier studies by other investigators of the effects of right temporal lobe damage and on the basis of results in this patient, that the callosal pathway from the right to the left temporal lobes must be taken into account in any theory of dichotic verbal tasks or of summation of binaural verbal information. It is tentatively suggested that the callosal pathway may normally be more important in both dichotic listening tasks with dissimilar stimuli and in tasks requiring summation than the ipsilateral pathway from the left ear. It is further suggested that the normally poor performance of the ipsilateral pathway may improve after damage to the corpus callosum or the right temporal lobe, especially after repeated exposure to such tasks.

It is the purpose of this paper to present studies of dichotic listening by a patient in whom Dr. P. J. Vogel had carried out a complete section of the neocortical commissures. We wish to thank Drs. Joseph Bogen, Roger Sperry and Michael Gazzaniga through whose kind cooperation this case was made available for study.

Physiological studies in animals (Tunturi, 1946; Rosenzweig, 1951) have shown that the projection from each ear to the contralateral temporal lobe is greater than to the ipsilateral temporal lobe. Bocca *et al.* (1955) were able to demonstrate superiority of the ipsilateral ear in patients with temporal lobe lesions. Broadbent (1954) and Broadbent and Gregory (1964) described the novel technique, now generally referred to as dichotic listening, in which different words are presented simultaneously to the two ears, thus producing auditory conflict. In an important series of papers Kimura (1961a, 1961b, 1967) and Milner (1962) have reported application of this technique to both normals and patients undergoing cortical excisions for epilepsy. They have shown a superiority of the right ear over the left by left-brain dominant normals in the task of reporting simultaneously presented digits. They report that following left temporal excisions in a patient population which is left-brain dominant the right

ear shows a significant degree of extinction in this task. Similarly, some degree of extinction by the left ear is seen after right temporal excisions. We have recently applied this technique to a group of aphasic patients with left hemisphere lesions as well as to a group of patients with right-hemisphere lesions without aphasia. These results will be published at a later date, but reference will be made to some of them in this paper.

This report will be devoted primarily to the results of examining dichotic listening in a patient with complete section of the neocortical commissures. Since this is primarily concerned with a single case, any conclusions drawn must of necessity be tentative. We feel, however, that the results are of sufficient interest and we record them in the hope that others with access to a larger group of such patients will be stimulated to further investigate this problem.

The patient, WJ, is a 52-year-old male who underwent complete section of his neocortical commissures by Dr. P. J. Vogel in 1962 for relief of seizures. He was the first patient in the series reported by Bogen and Vogel (1962) and Gazzaniga *et al.* (1963) who have given extensive reports of their findings. At operation the surgeon noted some atrophy of the right frontal lobe. The right frontal and occipital lobes were retracted at operation and the patient had a transient left hemiplegia post-operatively. At the Boston Veterans Administration Hospital where the patient was studied from September 9, 1966 to October 13, 1966, Dr. Ira Sherwin reported the following EEG findings:

> Prominent slow activity on the right side with a poorly defined slow wave focus in the right central parietal region, extending into the mid and posterior temporal region on the same side. In the same region there were long runs of excessively sharp activity with phase reversal tending to occur in the central parietal region. On hyperventilation considerable frontal slowing occurred bilaterally, especially on the right side.

The only elementary abnormalities observed in neurological examination at the Boston Veterans Administration Hospital consisted of some slowing in alternating movements of the left hand and on the left heel-shin test. Extensive signs of callosal disconnection were present. Comprehensive language examination revealed no evidence of aphasia. The Wechsler Adult Intelligence Scale, administered by Mrs. Edith Kaplan, indicated a normal Verbal I.Q. of 106, Performance I.Q. of 78, and a Full Scale I.Q. of 93.

I. PROCEDURE

The following tests were administered. Elementary base-line audiometric tests included pure tone audiometry using a standard audiometer, the SISI (*Short Increment Sensitivity Index*), Bekesy Audiometry, and the C.I.D. W-22 speech discrimination test.

Monaural tests preliminary to the dichotic hearing tests used the same list of digits and words as were used in the dichotic tests, but in different sequential order. The 20 digits presented alternately to each ear and the 20 names of animals presented in the same way are listed in Table I. Signal strength was SL + 44 dB (44 dB above sensation level for each ear).

Dichotic hearing tests included simultaneous presentation of two different lists of 20 single digits to each ear and simultaneous presentation of two different lists of animal names to each ear. Signal strength was again SL + 44 dB.

TABLE I

Dichotic tests: digit and word lists

Digits		Words	
List 1	List 2	List 1	List 2
2	6	horse	snake
9	4	bird	wolf
1	8	cow	fox
7	5	goat	dog
5	3	sheep	crow
8	2	cat	toad
3	10	lamb	rat
10	7	frog	mule
6	1	mouse	fish
4	9	bull	pig
8	1	wolf	bird
3	5	mule	frog
7	10	snake	horse
6	2	crow	sheep
9	4	rat	lamb
1	6	fish	mouse
2	8	dog	goat
10	3	pig	bull
5	7	toad	cat
4	9	fox	cow

The above tests were used not only with this patient, but also with a left-brain damaged aphasic and right-brain damaged non-aphasic population. Other tests designed to investigate possible variations in the degree of auditory extinction were used with our patient. One involved presentation of 25 infrequently used words to the right ear simultaneously with 25 frequently used words to the left. As in previous tests, signal strength was SL + 44 dB. Measurements of word frequency were determined by the Thorndike-Lorge Word List. The two lists are given in Table II. Dr. Melvin Barton rendered assistance in the design of this test.

The second additional test involved first, presentation of digits to the left ear and white noise to the right, and secondly, digits again to the left and unintelligible cocktail party babble to the right. The list of digits used for the dichotic listening tests (Table I) was again used in rearranged order. Signal strength was again at SL + 44 dB.

The third additional test again involved the rearranged dichotic listening digit test material, but with varying degrees of distortion by a lowpass filter of the list presented to the right ear. The left ear list was not distorted. Details will be given in the next section.

The fourth and final procedure was a summation test which involved presentation of incomplete information to each ear, also described in further detail in the next section.

TABLE II

Dichotic test: frequent and infrequent words

Left ear (frequent words)		Right ear (infrequent words)	
1. cut	13. ask	1. aft	14. nave
2. peace	14. dark	2. bare	15. pith
3. turn	15. chop	3. bode	16. quell
4. nose	16. gave	4. churl	17. trice
5. joy	17. burn	5. dolt	18. rife
6. rose	18. south	6. eke	19. sate
7. inch	19. fly	7. fife	20. vie
8. but	20. run	8. gibe	21. waif
9. else	21. come	9. hemp	22. squab
10. quite	22. page	10. irk	23. thwart
11. step	23. house	11. jaunt	24. loth
12. four	24. meat	12. larch	25. pap
	25. love	13. mauve	

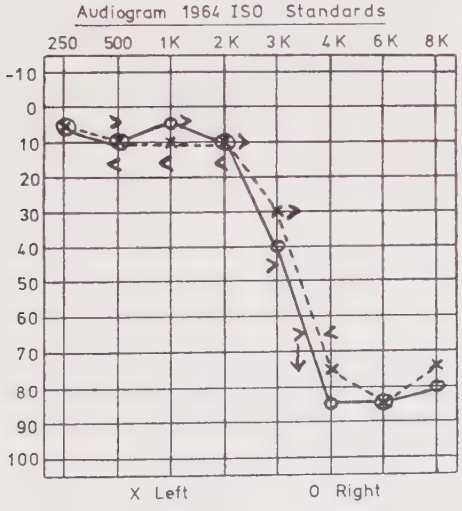
II. RESULTS

Pure tone audiometric examination (Figure 1) showed normal hearing in the speech frequencies for both ears, but marked bilateral elevation of thresholds above 3000 Hz. The SISI showed abnormal sensitivity to 1 dB increments at 4000 Hz only. Bekesy audiometry gave Type I tracings in both ears and showed the same thresholds as the standard pure tone audiometric examination. Speech discrimination scores were normal, 98% in the left ear and 94% in the right. They gave further confirmation of normal hearing for speech.

The dichotic hearing test involved transmission of the simultaneous signals from a two-channel tape recorder to a two-channel speech audiometer where calibration controlled the signal strength deviation to a variation of ± 2 dB.

The results of the dichotic presentation of digits and words to the 14 right-brain damaged patients and to WJ are shown in Table III. On the monaural preliminary test WJ scored 100% for each ear on both tests.

On the dichotic presentation of single digits or animal names WJ showed 100% extinction of the left ear. In other words, he was unable to repeat any digits or animal names presented to the left ear. Right ear per-



Speech Audiometry

	Left	Right
Thresholds:	10db	8db
Discrim:	98%	94%

SISI Scores

	Left	Right
Low Freq.	0%	0%
Mid Freq.	10%	5%
High Freq.	85%	90%

Bekesy Audiogram

	Left	Right
Type:	I	I

Fig. 1.

formance yielded perfect scores. Of fourteen right-brain damaged patients none showed 100% extinction by the left ear on the single digits test and only one showed 100% extinction on the animal names test. Details of the performance by the right-brain damaged group are shown in Table III.

TABLE III

Left ear extinction, dichotic listening tests

Digits	
Right-brain damaged patients:	
Number of patients	Degree of extinction
5	0%
4	30% or less
2	31%-50%
2	51%-80%
1	94%
Patient WJ:	100%

Words	
Right-brain damaged patients:	
number of patients	Degree of extinction
2	0%
4	30% or less
2	31%-50%
5	51%-90%
1	100%
Patient WJ:	100%

The above dichotic hearing tests were repeated on WJ one week later and again showed 100% extinction by the left ear. In a third re-rest immediately following the second, the patient was given stronger instructions to attend to the left ear and now scored 65% extinction by the left ear.

On presentation of common words to the left ear and uncommon words to the right WJ continued to show complete extinction of the left ear. Of the words presented to the right ear 44% were repeated correctly, 44% were repeated inaccurately (although recognizable similar to the stimulus words) and 12% were not repeated.

The patient performed normally on the test involving two types of

masking, achieving a 100% correct score with white noise masking in the right ear and a 95% correct score when cocktail party babble was given to the right ear. The patient recognized the presence of noise on the first test and of confused speech on the second in the right ear.

The results of the test involving simultaneous digits with the right signal distorted are of interest. It will be noted (Table IV) that as distortion in the right ear decreased, the correct responses from the left ear decreased and the correct responses from the right ear increased, giving evidence of increasing extinction of the left ear.

The summation test used methods similar to those described by Bocca *et al.* (1955), Calearo and Antonelli (1963), Jerger (1960) and Linden (1964). A list of twenty-two object names was used (Table V). An object name was presented to one ear at a sound level which in preliminary testing led to a score of approximately 50%. The other ear received the same word at SL + 44 dB, but distorted by a lowpass filter so that frequencies above a level of approximately 300 Hz were filtered out of the signal. Note that on this test the subject receives the same word in each ear, in contrast to the dichotic tests in which different words are given to the two ears. The summation tests explore the ability of the subject to integrate the partial information from each ear. Results are shown in Table VI. Scores for both ears were normal for monaural presentation of

TABLE IV

Dichotic test, varying distortion of right ear signal

Degree of filtering, right ear signal	% Correct	
	Left	Right
Low pass 540 Hz	90%	15%
Low pass 780 Hz	90%	10%
Low pass 900 Hz	50%	70%
Low pass 1020 Hz	20%	100%

On this test the right ear received only frequencies below the numbers indicated, so that maximum distortion was obtained with the 540 Hz low pass filtering and the least distortion with the 1020 Hz low pass filtering. In each case the left ear signal was undistorted. Signal to both ears was at SL + +44 dB.

TABLE V

Summation test word lists

Monaural (SL + 44 dB)					
Left			Right		
mice	bear	smile	dice	bread	bike
star	spears	bee	king	hat	queen
ring	rose	sew	pie	hose	toad
door	mail	dig	nun	sail	sea
well	rat	knot	wire	clocks	doll
chest	dart	vase	nest	bead	hot
tie	box	towel	book	tack	oar
man	chief	mop	can	leg	log
		kite	pear		
Monaural filtered (SL + 44 dB)					
Left			Right		
ice	deer	file	doll	wire	can
store	cart	towel	eye	road	bed
jar	bat	bee	dice	screen	tack
man	mop	fight	nun	pear	locks
vest	locks	lace	cane	hook	hat
sail	dig	rose	sea	leg	pail
bear	thief	saw	spike	beard	hose
		net	nest		
Filtered to left, weak normal to right			Filtered to right, weak normal to left		
mitt	pine	shop	car	owl	chop
tea	cat	slide	hog	dice	ax
wing	ham	sack	stair	tree	bat
socks	jail	boy	pearls	witch	seal
check	fire	key	pen	pliers	dart
block	war	ear	fly	clothes	crow
paw	pot	ship	fan	tea	bed
		hoe	bell		

normal signals at 44 dB above sensation level. The left ear did much more poorly than the right on filtered speech presented monaurally. On the summation tests, performance improved when filtered speech to the left ear was accompanied by a weak normal signal to the right. There was no improvement when filtered speech to the right ear was accompanied by a

TABLE VI

Summation test results

Monaural normal signal (SL + 44 dB)	Left 100%	Right 95%
Weak monaural normal signal	Left 50%	Right 50%
Monaural filtered signal (SL + 44 dB)	Left 23%	Right 68%
Filtered signal to left, weak normal to right	55%	
Filtered signal to right, weak normal to left	64%	

The first three lines are results of monaural tests. The last two are results of summation tests.

weak normal signal to the left. These results will be commented on in the discussion.

III. DISCUSSION

Let us first consider the patient's complete extinction of the left ear in the early testing when either digits or animal names were presented dichotically. It might be tempting to hypothesize that this was the result of damage to the right hemisphere. The patient had right frontal atrophy, as observed at operation. He was operated on the right side and had a significant right sided EEG abnormality. It should be noted that in the Montreal cases Milner (1962) found that frontal lobectomy did not lead to extinction, and right temporal lobectomy produced, on the average, only a small degree of extinction in the left ear. Data on our patients with right hemisphere lesions show a wide range of scores, but none did as poorly as WJ (Table III).

While we cannot exclude right-hemisphere damage as cause for this patient's extinction of verbal signals received by the left ear, we wish tentatively to offer another hypothesis, the correctness of which will depend on confirmation of these results in other patients with callosal lesions, but without right-hemisphere damage.

It has been customary in discussion of right versus left ear performance in dichotic listening tests to stress that each temporal lobe receives a contralateral projection and a smaller ipsilateral projection. We would like to add that material from one ear has two pathways for reaching the ipsilateral temporal lobe. One route is the direct one via the ipsilateral auditory pathway. Another route which is longer follows the stronger

contralateral pathway to the opposite temporal lobe and then returns via the corpus callosum to the ipsilateral temporal lobe. This callosal pathway has generally been neglected in theoretical discussions of dichotic listening in normals and patients with brain lesions. It would seem reasonable that the callosal pathway always plays a role in the left ear performance on dichotic listening tasks when the left temporal lobe is dominant since, as has been noted, left ear efficiency on this task declines after right

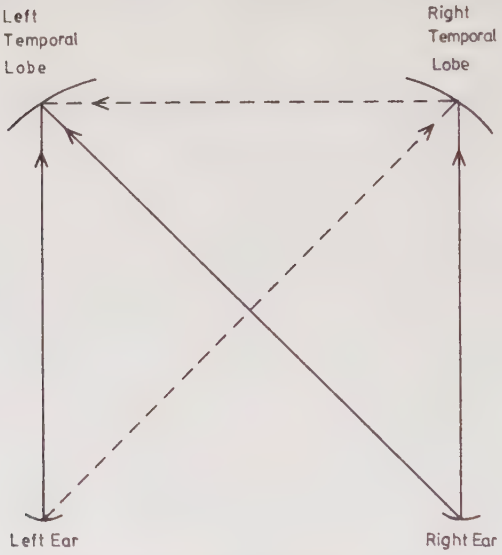


Fig. 2. Diagrammatic representation of auditory pathways. The broken lines represent the indirect (but probably more important) route from the left ear to the dominant left temporal lobe.

temporal lobectomy (Milner, 1962). Since verbal material to be reported must almost certainly reach the left hemisphere for decoding, the only way in which efficiency of the left ear could decline after right-temporal damage would be under conditions that the callosal pathway played some role in this task and that the weaker ipsilateral pathway could not be the only normal route to the dominant left temporal lobe. The existence in animals of a callosal pathway for auditory stimuli has been documented physiologically (Bremer *et al.*, 1956).

The results of the study of our patient would suggest the even stronger hypothesis that the callosal pathway is normally the most important one for reporting verbal material presented to the left ear in dichotic tasks. This would be compatible with the 100% extinction observed in our patient on this task. As Kimura (1967) has pointed out, the studies of Tunturi (1946) and Rosenzweig (1951) show that responses from the ipsilateral and contralateral ears after reception by one auditory cortex do not differ in latency or duration but only in amplitude. Since the callosal pathway would involve one extra synapse it is possible to speculate that the later arrival of verbal material at the left temporal lobe from the left ear via this callosal route might make it easier to resolve from material being received by the left temporal lobe from the right ear.

It should be noted that on repeated retesting of the dichotic digits task, WJ's left ear score improved from 0% detection (100% extinction) to 35% detection (65% extinction). This suggests that by specific practice the left temporal lobe may eventually begin to separate out messages coming via the weaker ipsilateral pathway in dichotic listening, although it normally inhibits them in this competitive situation.

Certain questions must arise concerning the above interpretation. Clearly, lesions of the cortical regions receiving the contralateral pathway from the left ear should produce extinction, as of course they do. But then the question arises as to why the degree of extinction by this patient is greater than that of our right hemisphere lesion cases and far greater than the average results cited, for example, by Milner (1962). There are two possible explanations. In the first place most of the hemispheric lesions will not be as complete as the disconnecting callocal lesions of WJ, all of whose interhemispheric connections were severed. Furthermore, in the Montreal material all the cases had lesions which had existed for some time before coming to surgery. Specifically, Milner (1962) states that these Montreal cases had "static atrophic lesions dating from birth or childhood." Hence, compensation by the ipsilateral pathway which was developed over a long period of time must be considered as a possibility to account for the average mildness of the effects after cortical excision for epilepsy.

One further implication of the above results must be mentioned. In the Montreal cases right-temporal lesions led to extinction in the left ear, and left-temporal lesions to extinction in the right ear. If the callosum plays

the role we have here hypothesized then the possibility must be considered that deep lesions involving white matter alone might produce similar effects. For example, a deep lesion in the left hemisphere involving callosal fibers from the right side might lead to extinction of verbal material in the left ear. Its effects would be the same as a lesion in the mid-callosum or a lesion involving the right temporal cortex or callosal fibers from that region. Our own data are at least consistent with this possibility since 35% of our right handed patients with aphasia and left hemisphere lesions have shown extinction of verbal material in the left ear rather than the right, while none of our cases with right hemisphere lesions have shown extinction of verbal material in the right ear. Such cases would not be expected after temporal excisions for epilepsy.

Some brief comment is necessary regarding the other tests. The suppression of the ipsiplateral pathway to the left temporal lobe is not complete since white noise and even indistinguishable babble produced by multiple voices presented to the right ear did not lead to extinction by the left ear. This suggests that suppression of the left ear signal will occur only if there is a marked similarity in the signals reaching the left temporal lobe from the two ears. When this similarity is marked the left ear fails totally. Thus, even when we attempted to 'help' the left ear by giving it common words while the right ear received uncommon words the extinction score was still 100% on the left.

This effect of similarity is further borne out by the dichotic test in which the right ear received distorted speech while the left ear received normal signals, both well above thresholds. As the right ear score improved with decreasing distortion, the left ear score reflected increasing suppression.

The left ear did more poorly than the right in monaural presentation of filtered speech when summation was not required. Sinha (1959) showed that perception declined in the ear contralateral to a temporal lobe lesion when signals which were distorted by the addition of masking were used. Basing our opinion on the earlier discussion, we would expect a callosal lesion to produce a disturbance similar to that seen after right-temporal lobectomy, a supposition borne out by WJ's performance when presented with distorted speech to the left ear.

The third and fourth lines of Table VI present the scores for the summation tasks. This test differs from dichotic listening tests since the same word is presented to both ears and the task involves summation of partial

information from both ears. In normals the score on this task is higher than either monaural score, the improvement reflecting the process of summation.

WJ's performance, on the other hand, shows no indication that integration of the information from the two ears occurred. When the left ear received the filtered signal and the weak but normal signal went to the right ear the 58% score suggests that responses were based on the weak but normal signal received by the right ear, previously determined as being approximately 50%. The impression gained is that transfer of attention to the right ear with its functioning contralateral projection took place. The same lack of summation occurred when the above procedure was reversed so that the right ear received the filtered signal and the left received the weak normal. The score was reflective of that achieved by the right ear for filtered speech alone. Limitation of time prevented more intensive study of this procedure to see if repeated testing might produce any evidence of summation. We feel that this problem deserves further study to test the preliminary hypothesis that summation normally requires an intact callosal pathway.*

Although any final conclusions must be tentative we feel that the participation of the callosum in dichotic listening tasks must be postulated to account for decline in left ear scores on dichotic listening tests following right-temporal lobectomy. The results from this patient suggest the even stronger working hypothesis that the callosal pathway from the right to left temporal lobe may be more important than the ipsilateral auditory pathway from the left ear in dichotic listening tasks in normals, and that the performance of the ipsilateral pathway from the left ear may improve from its initially lower level after callosal section or damage to the right temporal region, particularly if special training is given.

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NOTES

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CHAPTER XVI

ISOLATION OF THE SPEECH AREA

PREFACE

The credit for discovering this remarkable patient and for studying her so carefully over many years goes to Dr. Fred Quadfasel, who also demonstrated her remarkable capacity for verbal learning. Without these astute clinical and experimental observations the subsequent post-mortem findings would have been of little use. Combining the two, however, made it possible to draw far-reaching theoretical conclusions from this experiment of nature. Perhaps the most important conclusion is that comprehension of language is not unitary. Thus this patient could complete common phrases, and could perform other discriminatory activities, while she failed at others. This conclusion is reinforced by other findings, e.g., some patients with severe comprehension deficits for questions and commands for the individual limbs may yet respond very well to commands carried out with the axial musculature, e.g., 'sit down', 'stand up' 'walk', etc.

Although repetition disorder of the type presented by this patient had been described before, I believe that the remarkable preservation of verbal learning in the presence of a severe acquired comprehension deficit had not previously been described. The explanation of this phenomenon as given in the paper fits in well with many of the same anatomical findings which account for the preservation of repetition.

ISOLATION OF THE SPEECH AREA*

1968

ABSTRACT. The case is presented of a 22-year-old woman who survived ten years following an episode of carbon-monoxide poisoning. The patient's language behavior presented several striking clinical features. Her spontaneous speech was confined to a few stereotyped phrases and she gave no evidence of comprehension of language. By contrast she exhibited echolalic repetition with excellent articulation. In many instances she showed the 'completion' phenomenon described by Stengel, i.e. the completion of stereotyped or simple phrases uttered by the examiner. In addition she was able to carry on verbal learning as shown by her ability to learn the words (as well as music) of songs which had not existed before her illness.

At post-mortem, a detailed study of serial whole-brain sections showed intactness of auditory pathways up to and including Heschl's gyrus, of Wernicke's area and Broca's area and of the arcuate fasciculus connecting these two cortical regions, of the lower Rolandic cortex and of corresponding portions of the pyramidal tract. The hippocampal region (except for Sommer's sector) and the structures of the limbic system were well preserved, as was the reticular substance of the brain stem.

This case corresponds in its clinical picture to the classical 'mixed transcortical aphasia' (i.e. paucity of speech and severe comprehension deficits with excellent preservation of repetition) with some new features (capacity for verbal learning) not previously described. The pathology agrees with that advanced by Goldstein for this condition, i.e. 'isolation of the speech area'. The explanation advanced for this clinical picture is that comprehension and propositional speech are lost because of the isolation of the speech region from other cortical areas, while those functions which can be carried on by the speech area itself, i.e. repetition and completion of well-learned phrases, are preserved. The ability to carry on verbal learning in this patient probably depended on the intactness of the medial temporal regions and the preservation of the connections to them from Wernicke's area.

The term 'transcortical aphasia' has been little used in recent years although in an earlier period it served to define a group of syndromes of great interest. Goldstein (1917) summarized the literature in a lengthy monograph and gave his own extensive interpretations of this group of syndromes. Further discussions also appear in his later writings (Goldstein, 1927, 1948). He pointed out that the term was coined by Wernicke, and it came to be used for those syndromes in which repetition is well preserved in spite of marked diminution of spontaneous speech (transcortical motor aphasia), or gross impairment of comprehension (transcortical sensory aphasia), or of both (mixed transcortical aphasia). Recent writings on aphasia

devote little space to these syndromes. Thus Weisenburg and McBride (1935) and Brain (1961) mention it in a few lines, and inaccurately summarize Goldstein's views. Echolalia, i.e. forced repetition, which may be a feature of these syndromes, has been treated by Stengel (1936, 1947) who gives an excellent discussion of the clinical features of this phenomenon. Hécaen *et al.* (1967) present a detailed linguistic analysis of a case in whom echolalia was prominent and was thought to reflect the perseverative aspects of disturbed attention.

Goldstein (1917) discusses a wide variety of transcortical syndromes, but we will confine ourselves to the group most relevant to the case presented below, i.e. those in which repetition is excellent despite severe impairment of comprehension, i.e. the sensory and mixed transcortical aphasias. He summarizes three possible mechanisms for this type of transcortical aphasia:

(1) It might result from a mild lesion of Wernicke's area (i.e. the posterior part of the first temporal gyrus) or as a stage in the recovery from Wernicke's aphasia, a possibility suggested by several other authors. Goldstein did not emphasize this mechanism since he thought that patients falling in this group would not show excellent repetition although repetition might be somewhat better than comprehension. He thought that many supposed cases of this type were actually instances of the third mechanism listed below partially masked by a mild involvement of Wernicke's area.

(2) With marked destruction of Wernicke's area the patient would usually show severe loss of comprehension but might show excellent repetition if this function were mediated through the right hemisphere. Goldstein thought that this mechanism was infrequent because he believed that only rarely could the right hemisphere take over the function of repetition. Stengel (1947) states that the case reported in his earlier paper (1936) was found at post-mortem to have "a thrombosis of the left middle cerebral artery – which had resulted in destruction of the whole speech area." No more detailed description of the anatomy has been published, but if this description is correct this case would have to be regarded as one in which repetition was carried on by the right hemisphere. Goldstein did not regard these two mechanisms as very important in the pathogenesis of this syndrome. They are the only ones mentioned by Weisenburg and McBride (1935) and Brain (1961) who omitted

to mention the third mechanism upon which Goldstein placed greatest emphasis.

(3) The third mechanism was called 'isolation of the speech area' by Goldstein. In this case the entire classical speech area, i.e. Wernicke's area and Broca's area as well as the connections between them, are spared. Goldstein's discussion also implies that afferent auditory pathways to the left hemisphere and the input to Wernicke's area from the left Heschl's gyrus are spared, as well as the output from Broca's area. The main feature of the lesion is that it disconnects Wernicke's area from the posterior 'ideational field' (Begriffsfeld), i.e. the posterior parietal association cortex. The patient can repeat because of the intactness of the speech areas, but does not comprehend because Wernicke's area cannot arouse associations in other parts of the brain. Similarly, what comes into other portions of the brain cannot reach Wernicke's area and thus the patient does not produce normal speech. Some patients with this pattern of lesions produce fluent, grossly paraphasic speech, while others produce little spontaneous speech. Goldstein thought that for the production of fully developed echolalia it was necessary to have a lesion of the frontal 'ideational field', i.e. of those parts of the frontal lobes lying anterior to Broca's area, whose destruction would lead to a loss of spontaneous speech together with a heightened degree of uninhibited repetition. This corresponds with the observation that frontal lesions may cause a loss of spontaneous activity although the immediate response to stimulation may be increased.

It should be noted that Goldstein's third mechanism is similar to that proposed earlier in less elaborate form by Lichtheim (1885) who thought that the transcortical aphasias were due to lesions between the motor or sensory speech areas and those parts of the brain important for non-language mental functions.

We wish to present in this paper a patient whose clinical picture and post-mortem findings appear to correspond closely to the syndrome of the 'isolated speech area' as presented by Goldstein. This report also presents a phenomenon not previously described to our knowledge in a case of echolalia without comprehension coming on in adult life, i.e. the ability to carry on verbal learning. Some of the findings have been mentioned briefly in earlier publications (Segarra and Quadfasel, 1961; Geschwind, 1965).

I. CASE HISTORY

The patient was a 22-year-old woman admitted to the Cushing Veterans Administration Hospital on September 6, 1950. On October 7, 1949, she was found apneic in the kitchen of her home with the unlit gas jet of the hot water heater turned on. Artificial respiration was given and breathing restored. She was sent first to a local hospital and then to a naval hospital where she remained in coma for seventeen days. When she recovered consciousness her examiners noted that she sang songs and repeated questions. On several occasions when the examiner said, "Ask me no questions" she would, instead of repeating, reply, "I'll tell you no lies." While in this hospital she had seizures involving the right and left sides of the body at different times. She was transferred to the Cushing Veterans Administration Hospital in September, 1950 and remained there and later at the Boston Veterans Administration Hospital until her death on August 19, 1959. She was followed continuously by one of us (F.A.Q.).

On admission to the Cushing VA Hospital all relevant abnormalities were in the neurological examination. She lay in bed following the examiner's movements with her eyes. When asked questions she would repeat them again and again. Her spontaneous speech was confined to the phrase 'Hi, daddy" reiterated several times or some unrecognizable words. She followed no commands. She would grasp the examiner's hand or some other object strongly and would not release it. She might display fear or marked agitation on stimulation.

Visual fields could be tested only roughly, but she responded to movement in all quadrants. The optic fundi were normal. Ocular and lid movements were normal, as were pupillary responses and corneal reflexes. The motor components of the fifth nerve were intact and facial sensation seemed normal. Facial movements were normal. Hearing seemed normal (as evidenced by patient's ability to repeat words spoken to her). The gag reflex was present, swallowing and phonation were normal. The tongue was moved spontaneously in all directions.

The patient made few spontaneous movements except the grasping of objects mentioned above. When disturbed she would move her arms about and make repetitive movements such as stroking her hair. She resisted passive movements of the arms. Strength in the arms and the grip in particular appeared to be normal. Both legs were very spastic, and the only

movements seen were the return to the flexion position after passive extension. Deep tendon reflexes were hyperactive throughout, being somewhat greater on the left, and there was unsustained clonus in the left ankle. Abdominal reflexes were present and the plantar responses were flexor. Coordination appeared normal in the arms but could not be tested in the legs. The patient responded to pin prick everywhere with signs of displeasure, but no further sensory testing could be done. The patient was incontinent of urine and feces.

Routine laboratory and X-ray studies were normal, except for mild evidences of urinary tract infection. CSF was normal on all examinations. A pneumoencephalogram showed dilatation of the subarachnoid spaces and of the ventricles, particularly of both temporal horns. EEGs showed bilateral non-focal fast activity.

The patient's neurological state remained essentially unchanged. Special investigations of her ability to repeat and to learn songs will be given below. She would often show marked agitation and sometimes injured ward personnel. Right-sided seizures were controlled by diphenylhydantoin. Repeated urinary infections responded to antibiotics and tidal drainage.

In June, 1957 the patient underwent an amputation above the right knee for a non-healing fracture. At this time her verbal behavior began to diminish and by July, 1959 it was found that the patient had ceased spontaneous speech or repetition, but was otherwise unchanged. On August 19, 1959 the patient suddenly developed a high fever and coma and died in a few hours, presumably in septic shock. General postmortem examination showed evidence of mild pulmonary and urinary tract infection. The examination of the brain will be reported in detail in a later section.

II. SPEECH AND LANGUAGE STUDIES

The major interest of this patient was her speech and language behavior which remained essentially unchanged over most of her hospital course until the last two years before her death when she gradually ceased spontaneous speech or repetition. She was studied repeatedly by one of us (F.A.Q.) who was in charge of the patient during her entire stay at the Cushing and Boston Veterans Administration Hospitals.

The patient's spontaneous speech was limited to a few stereotyped phrases, such as, "Hi, daddy," "So can daddy," "Mother," or "Dirty bastard." She never uttered a sentence of propositional speech over the nine years of observation. She never asked for anything and she never replied to questions except in a certain special way to be mentioned below. Similarly, she showed no evidence of ever having comprehended anything said to her (with some special exceptions to be detailed below) either by her verbal responses or by execution of commands. Similarly, she never wrote nor responded to written material. As noted earlier, despite the presence of adequate movement in the arms, she never used them except in such acts as stroking her hair, grasping, or striking her attendants.

Despite the notable lack of spontaneous speech, it was noted that she generally repeated questions in a normal voice without dysarthria. Occasionally she would, instead of repeating a phrase, complete it in a conventional manner. Thus to, "Ask me no questions" she would at times reply, "Tell me no lies." Similarly she would sometimes complete proverbs or titles of songs. This is the 'completion' phenomenon described by Stengel (1936, 1947) and will be commented on in the discussion. A related phenomenon was her tendency to be triggered to produce certain phrases by words used by the examiner. Thus when told, "Close your eyes," she might say, "Go to sleep." When asked, "Is this a rose?" she might say, "Roses are red, violets are blue, sugar is sweet and so are you." To the word 'coffee' she sometimes said, "I love coffee, I love tea, I love the girls and the girls love me."

An even more striking phenomenon was observed early in the patient's illness. The patient would sing along with songs or musical commercials coming over the radio in her room or would recite prayers along with the priest during religious broadcasts. This ability was tested both formally by one of us (F.A.Q.) and informally by the nurses and other physicians caring for the patient. If a record of a familiar song was played the patient would sing along with it. If the record was stopped she would continue singing correctly both words and music for another few lines and then stop. If the examiner kept humming the tune without words, the patient would continue singing the words to the end. It was obvious that she could remember the words since she would sing them for a period without a model to imitate. It was therefore decided to find out if she could learn songs not previously known to her. It has been found that she would sing

musical commercials or popular songs that had not existed before her illness. New songs were played to her and it was found that after a few repetitions she could learn these as evidenced by her ability to sing a few lines correctly after the record had been stopped or to keep on singing if the examiner continued to hum the tune without the words. It was therefore obvious that the patient was not merely repeating the words produced by the record or the examiner. Furthermore, she could sing two different sets of words to the same melody, depending on which version was started by the examiner. For example, she could sing "Let me call you sweetheart" with the conventional words, but also learned a parody beginning "Let me call you rummy." Her articulation of the sounds and her production of melody were correct although she might sometimes substitute the words "Dirty bastard" for some of the syllables of the song, but this occurred irregularly.

III. NEUROPATHOLOGICAL FINDINGS

The whole brain, including cerebellum and brainstem, was fixed in 10% formalin and embedded in celloidin. Through the kindness of Dr. Paul Yakovlev serial coronal sections of the whole brain were made. Every 20th section was stained for myelin (Loyez) and an adjacent section stained for cells with cresyl violet; 3370 slides were obtained. One of us (J.M.S.) made a detailed mapping of the lesions. Subsequently a detailed review was made with Dr. Paul Yakovlev to confirm the correctness of the original mapping.

Histopathological Character of Lesions

The type of lesion was very similar throughout, and followed the well known pattern in cases of carbon monoxide poisoning. Extensive areas of cortex showed laminar necrosis especially prominent in the deep layers (5th and 6th) and the zone of transition to the white matter. This old, gliotic, ischemic necrosis would sometimes involve the full depth of the cortical ribbon so that no neural elements were left, the tissue being a pure gliotic scar.

Frequently, however, cortical ischemic necrosis was more focal taking the form of oval elongated regions of pallor (Erbleichungsherde) in which the neural population had disappeared, the glia had proliferated and the pial surface had sunk in to give the appearance known as granular

atrophy. Sommer's sector of the hippocampus showed nearly total cell loss (Figure 1).

Damage, however, was not confined to the cortex. The myelin sheaths of the subcortical white matter also suffered in some areas and rather large areas of myelin pallor could be seen in the centrum semiovale of the frontoparietal region. In some portions of the temporal lobes the myelin loss was nearly complete.

The globus pallidus suffered profoundly and of its anterior $\frac{1}{3}$ there remained only a spongy cyst. The posterior $\frac{2}{3}$ were preserved.

Topographical Distribution of Lesions: (Figures 1–9)

(*a*) *Frontal lobes.* The superior frontal convolution on the right side was severely damaged at the frontal pole and along the convexity, but the medial aspect was fairly well preserved. On the left side the cortex was also undermined by severe laminar necrosis and subcortical myelin loss.

The middle frontal convolution although dotted with numerous areas of cortical pallor and undercut by extensive laminar necrosis retained nevertheless a larger neuronal population than the superior gyrus.

The inferior frontal convolution on the right side was severely involved in some places; this was in contradistinction to the one on the left side which had most of its nerve cells still intact. Sections passing through the tips of the lateral ventricles show very well how both superior and middle frontal gyri were involved while the inferior convolutions were better preserved. In particular Broca's area appears nearly normal (Figure 2), but the corresponding region on the right is more involved.

The motor cortex showed maximum damage in its upper half especially in the 'leg area'. This was consistent with the paraplegia presented by the patient. The orbital cortex was severely damaged on the left but spared on the right.

(*b*) *Parietal lobes.* There was extensive damage in the upper half of the superior parietal and supramarginal gyri. The post-cental gyrus and both lips/of the intraparietal sulcus seem to have withstood better the onslaught of anoxia. The lower supramarginal and the angular gyri were well preserved on the left, while the right supramarginal gyrus was severely damaged.

(*c*) *Occipital lobes.* The pattern and extent of the lesions was similar in both occipital lobes. The convexity of the parieto-occipital cortex (posteri-

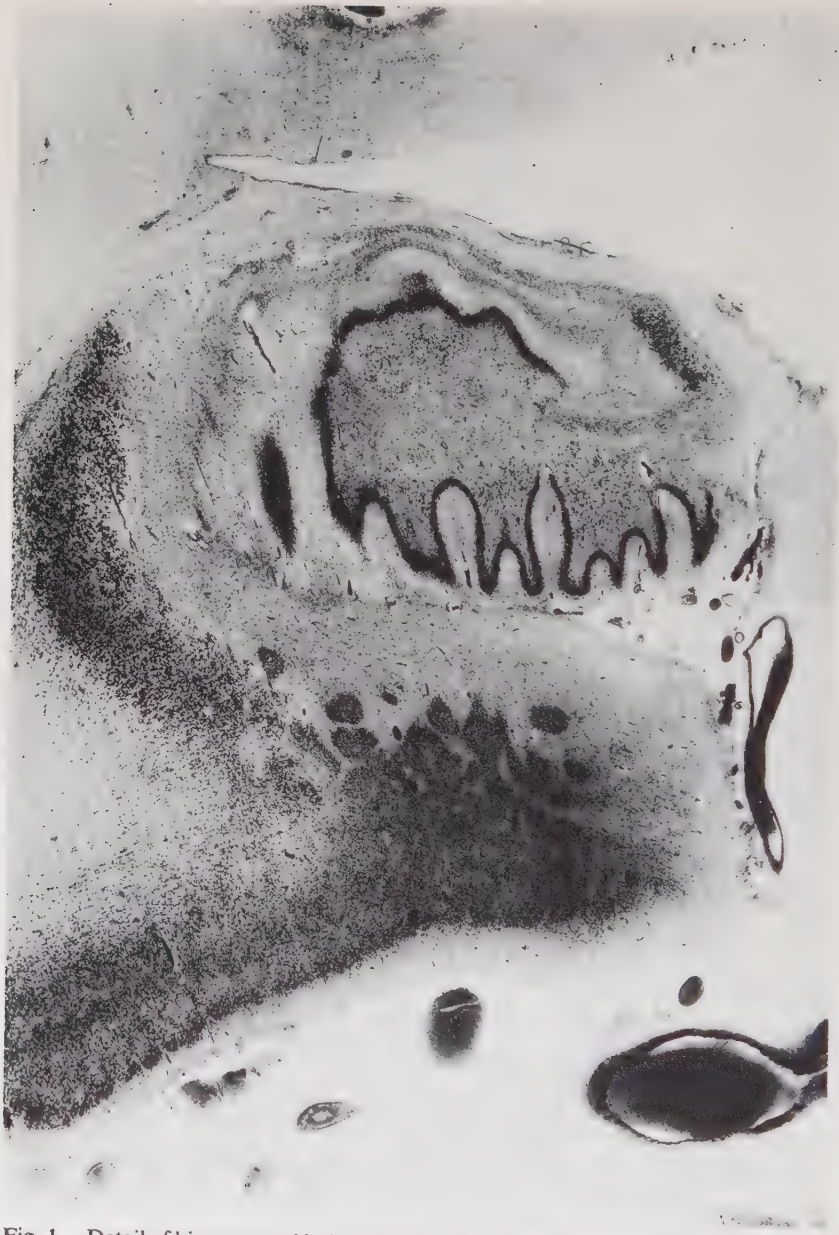

Fig. 1. Detail of hippocampal lesions. Nissl, 40 ×. Observe necrosis of Sommer sector with preservation of the remaining hippocampal cortex.

or parietal, anterior occipital gyri) as well as the undersurface of the hemisphere (fusiform gyrus) showed extensive cortical necrosis, either complete or laminar. The areas affected are PM, PG, OA and to a lesser degree PEP. The calcarine cortex was preserved throughout. The precuneus (PE) and the lingual lobe were also preserved. The most posterior area, before the very end of the occipital pole, was severely damaged. The subcortical white matter belonging to these damaged areas showed considerable loss of fibres, but the deeper periventricular strata and the fasciculus transversus occipitalis were preserved.

(*d*) *Temporal lobes.* The cortex of the tips of both temporal lobes was preserved especially in the lower portions corresponding to the tip of the second temporal convolution. However, the subcortical white matter

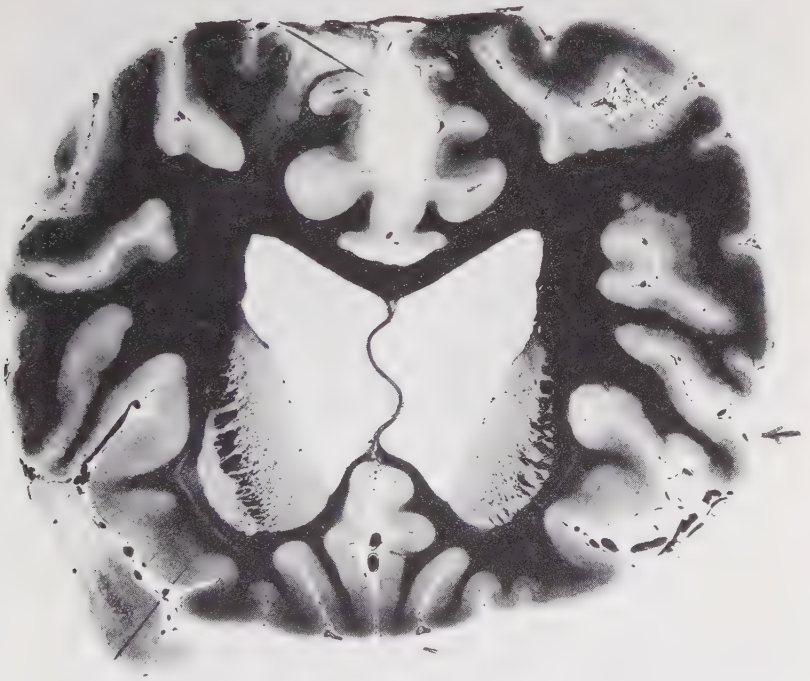

Fig. 2. Preservation of Broca's area (arrow) in the left hemisphere. Myelin.

was completely destroyed for about 4 to 6 cm beginning at the very tip.

The lesions were remarkably symmetrical. Area TG2 and the hippocampal enclave HA were preserved. Ischemic necrosis was more intense along the bottom of the parallel sulcus (first temporal sulcus) and the convexity of the second temporal convolution, (area TE of Von Economo, Brodman's 20 and 21). The opercular aspect of the first temporal gyrus as well as Heschl's transverse gyri were well preserved (Figure 3). Wernicke's area (i.e. the left posterior superior temporal gyrus) was thus preserved throughout.

In more caudally situated sections both temporal lobes were severely

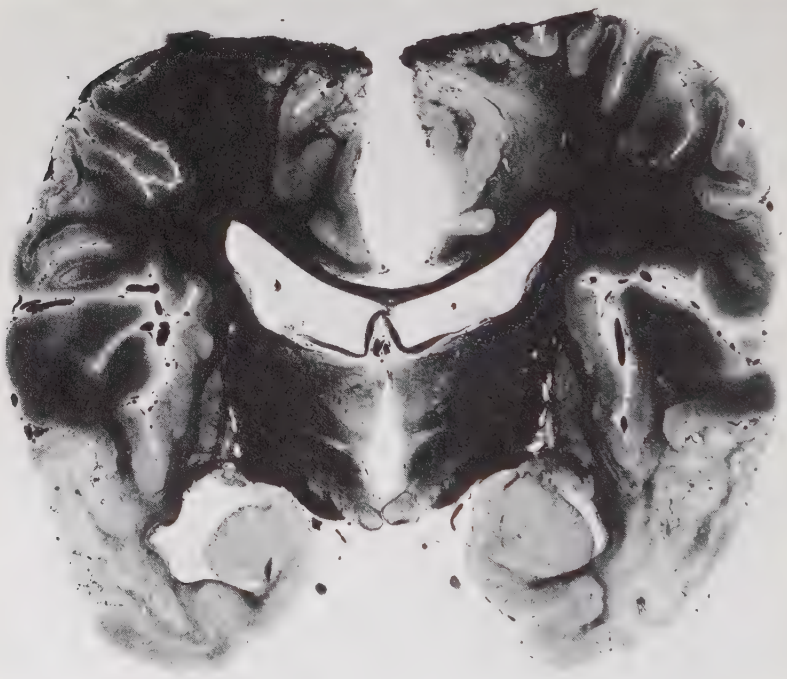

Fig. 3. Coronal section through thalamus. Myelin stain. Observe the preservation of primary auditory areas (Heschl's gyrus), Wernicke's area, insular cortex and lower perisylvian cortex. Arcuate fasciculus also preserved. Leg areas of motor-sensory cortex and remainder of temporal lobe convexities are severely damaged. Note marked thinning of corpus callosum.

damaged and their white matter wiped out except for the optic radiations and the inferior longitudinal fasciculus.

The parahippocampal area was well preserved. The hippocampus proper showed damage of the Sommer sector in a consistent manner, while elsewhere in the hippocampus proper, lesions were rather patchy and relatively moderate (Figure 1); there were some foci of destruction but they were not large and they left most of the layers of the fascia dentata in a fair degree of preservation. Moreover both fornices were well preserved, thus confirming the relative integrity of the hippocampi.

The connections of the anterior temporal region with the thalamus, the so-called pedunculus thalami extracapsularis running dorsal to the optic tract and parallel to the anterior commissure were relatively spared (Klingler and Gloor, 1960).

The amygdalar complex of nuclei and the substantia innominata of Reichert were spared. The temporopontine fasciculus of Türck was somewhat thin. The fasciculus amygdalo-temporalis, running from the amygdalar nuclei to the temporal pole, appeared degenerated. The anterior commissure and the fasciculus uncinatus were well preserved.

(*e*) *Cingulate gyri.* Both cingulate gyri were remarkably preserved in almost their entirety. There was a 4 mm length of involved cortex along the inferior lip of the callosomarginal sulcus, but otherwise the limbic cortex was spared.

(*f*) *Insula.* Both insulae were preserved except for a relatively moderate amount of deep laminar necrosis in some spots. The capsula extrema was spared, but the capsula externa showed some pallor on the right side.

(*g*) *Basal ganglia.* The well known necrosis of the globus pallidus was present. The anterior half of this structure was reduced to a cyst partly filled with a loose lacework of glial fibres. This involved both the inner and outer segments (Figure 4).

The putamen and the caudate nucleus had both lost many nerve cells and there was considerable gliosis in them. This was particularly true of the superior angle of the putamen.

(*h*) *Thalamus.* (1) Anterior nuclear group: The cell population of the nucleus anterior ventralis (AV) was only mildly involved; the thalamic tubercle stood out prominently in the lateral ventricles in both sides. The same findings applied to nuclei anterior medialis (AM) and anterior dorsalis (AD).

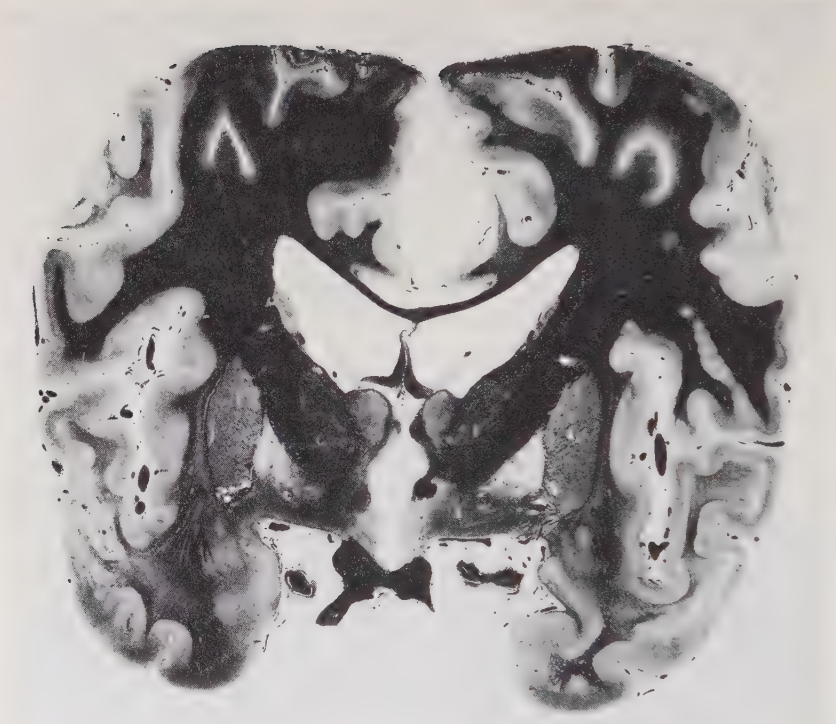

Fig. 4. Coronal section. Myelin stain. Observe cystic necrosis of globus pallidus on both sides. Note preservation of fornices.

(2) Nucleus ventralis anterior (VA): This nucleus was degenerated and many of its neurons were lost with a corresponding increase in glia.

(3) Nucleus ventralis lateralis (VL): Considerable diminuation was noted of its nerve cell population, with increase in glia and perhaps some pallor of the anterior thalamic radiations.

(4) Nucleus medialis dorsalis (MD): This nucleus had several irregular patches where neurons had disappeared. They were distributed around vessels and were probably the result of direct action of the ischemic process rather than of secondary degeneration. The medial half of the nucleus had more neurons preserved.

(5) Nucleus ventralis postero-lateralis (VPL) and ventralis postero-medialis (VPM): On the whole both nuclei on both sides were spared.

(6) Pulvinar: The lateral, and more so the medial, pulvinar were frankly gliotic even though the nerve cell loss was only moderate.

(*i*) *Cerebellum.* The cerebellar cortex was better preserved than expected. There was only moderate loss of Purkinje cells. The dentate and roof nuclei were also preserved.

(*j*) *Brain stem.* Except for pallor of the pyramidal tracts, the main nuclear formations and tracts were within normal limits. There was no evidence of involvement of any portion of the reticular formation.

Secondary Degenerations

(*a*) *Corpus callosum.* Taken as a whole it was evident that this fibre system was considerably thinned (Figures 3, 4) and reactive gliosis reflected the fibre loss. On myelin stains, however, it was hard to trace specifically degenerated fibres in relation to particular areas of cortex. The genu showed severe gliosis and a loss of fibres that could be grossly evaluated as 40 percent of the normal content. Even more impressive was the thinness of the body of the corpus callosum (from 1.5 to 2 mm in the coronal plane). This thinness was present all along the structure except for the splenium.

The callosal radiations and tapetal fibres were for the most part intact.

(*b*) *Anterior commissure.* There was no detectable lesion in it except for some diminuation of the total number of fibres.

(*c*) *Fornix.* On the whole the fornix and other circular fibre-systems (indusium griseum, terminal striae) were spared. The medial and lateral components of the fornix were both well myelinated, as were the columns and the pars tecta.

(*d*) *Superior longitudinal fasciculus.* Frontal and parietal white matter show myelin pallor on the right outside the corona radiata in the region corresponding to this fibre system. In the left hemisphere only relatively mild myelin loss could be detected in this general area.

(*e*) *Superior occipito-frontal fasciculus.* The typical 'braided' appearance of this association tract was preserved. There was, however, a difference between the two sides since the tract on the left was pale and gliotic and had lost a good number of fibres in comparison with its companion on the right.

(*f*) *Inferior longitudinal fasciculus.* Because of surrounding demyelination this fasciculus could be easily traced all the way to the occipital pole. It appeared normal on both sides.

(*g*) *Inferior occipito-fronto fasciculus.* By contrast, the left occipito-frontal tract had lost many fibres while the one on the right, though pale, was still within the limits of variation of the histological technique.
(*h*) *Cingulum.* Appeared intact on both sides at the frontal level. In more caudal sections there was definite fibre loss on the left side.

Fig. 5. Composite diagram of extent and nature of lesions as viewed from above. Dots represent cortical damage with more severe lesions indicated by greater density. Oblique lines represent sub-cortical white matter lesions. Observe the relative preservation of sensory-motor cortex, with the exception of leg areas.

(*i*) *Fasciculus uncinatus.* Although a fair number of fibres had disappeared on the left side there was definite preservation of at least 50 per cent of this structure. The fibres did not reach the very pole of the temporal lobes on either side but there were viable connections to and from the upper lip of the anterior portions of the first temporal convolution and the surviving frontal cortex. In the right hemisphere the main body of fibres was preserved.
(*j*) *Other fibre systems.* The fronto-pontine and the parieto-pontine contingents had lost most of their fibres. Similar though less intense pallor could be seen in the ansa lenticularis. The subcallosal fasciculus was

probably spared. The transverse occipito-parietal fasciculus was also within normal limits.

Summary of Anatomical Lesions

The cortical and subcortical white matter lesions described above in detail were plotted out on cortical maps of the hemisphere so that it is possible

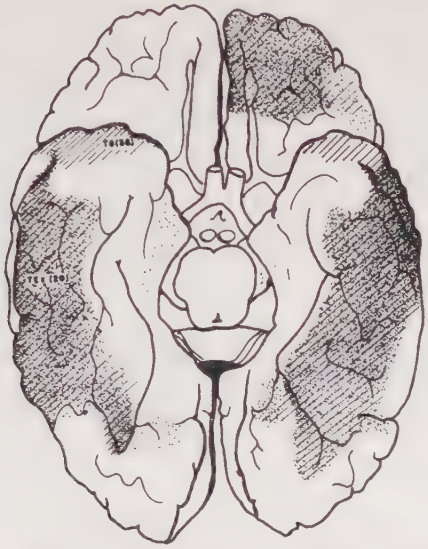

Fig. 6. Basal view. Observe the destruction of left orbital cortex and lower convexity of both temporal lobes. Both temporal poles show preserved cortex but the subcortical lesion completely isolates them. Hippocampal gyri show minimal lesions.

to see easily that certain areas of the brain were spared completely or nearly completely (Figures 5–9). The lesions are generally symmetrical in both hemispheres except for the markedly asymmetrical destruction of the left orbital cortex. The completely or generally preserved regions were as follows:

(a) Opercular, perisylvian cortex of frontal, parietal and temporal ret gions including the insula. This region was better preserved on the left- than on the right.

(b) Cingulate gyrus.

(c) The occipital lobe and adjacent medial parietal areas.

The first area corresponds roughly to the territory of the middle cerebral artery and on the left includes the classical speech areas, i.e. Wernicke's area, Broca's area and their interconnections via the arcuate fasciculus.

The second area involves the medial aspect of both hemispheres roughly corresponding to the classical 'grand circonvolution limbique' of Broca. With the isolated exception of ischemic change limited to the

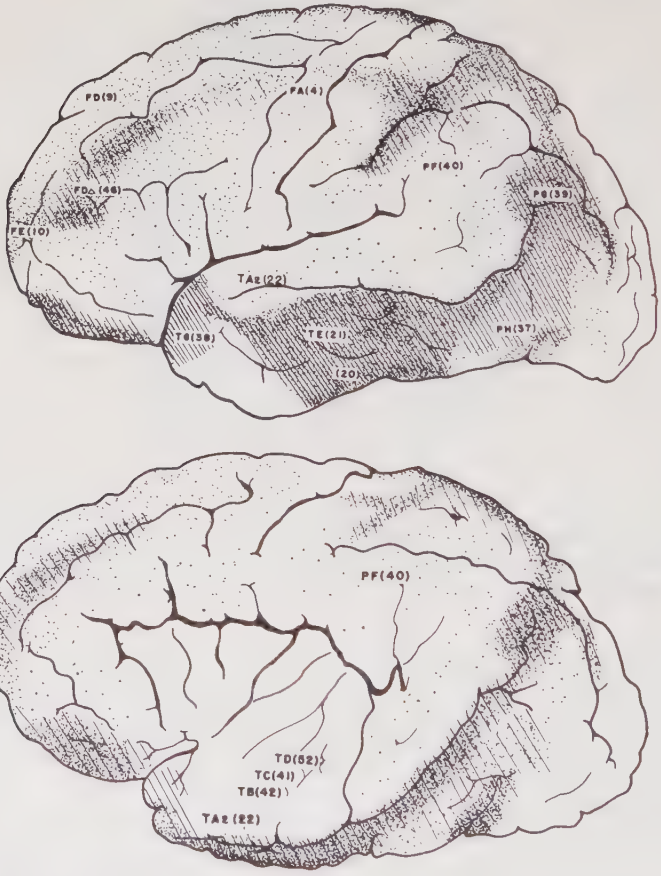

Fig. 7. Left hemisphere. The diagram below shows the insula with the lips of sylvian fissure widely separated. Observe the preservation of cortical structures related to language (Broca, Wernicke, arcuate fasciculus). The central area is surrounded by a large C-shaped lesion which isolates speech areas from the rest of the brain.

Sommer sector of the hippocampus one finds a remarkable preservation of the majority of the 'visceral brain' including not only the parahippocampal gyrus, the hippocampus proper, the fornix and the cingulate gyrus but the prepyriform and subcallosal cortex as well.

Finally there was preservation of both occipital poles and calcarine cortexes.

Bilateral necrosis of the globus pallidus is of course a classical finding

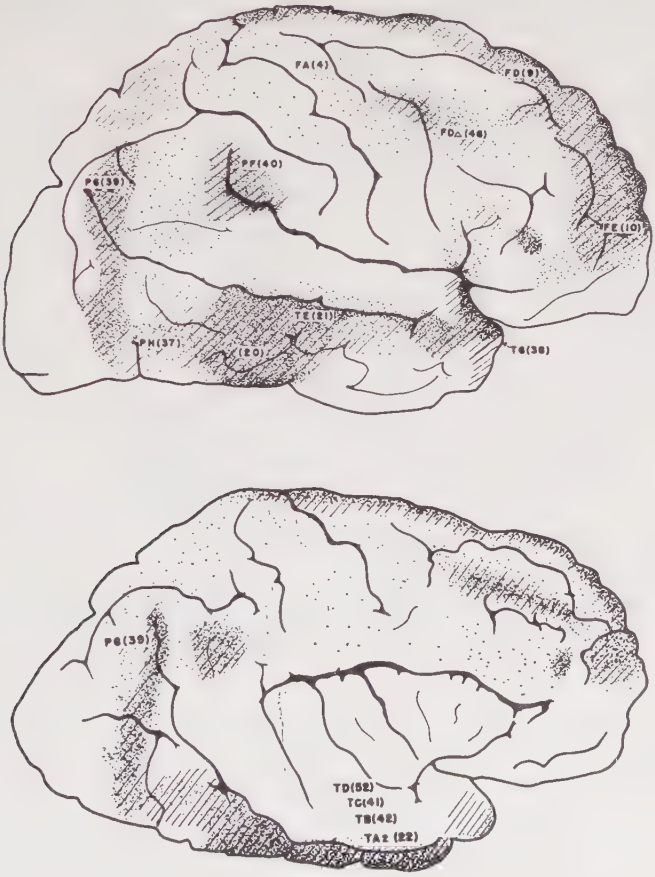

Fig. 8. Right hemisphere. Below, the sylvian fissure has been opened to view the insula. Observe the preservation of motor-sensory cortex, and the central part of the territory of middle cerebral artery.

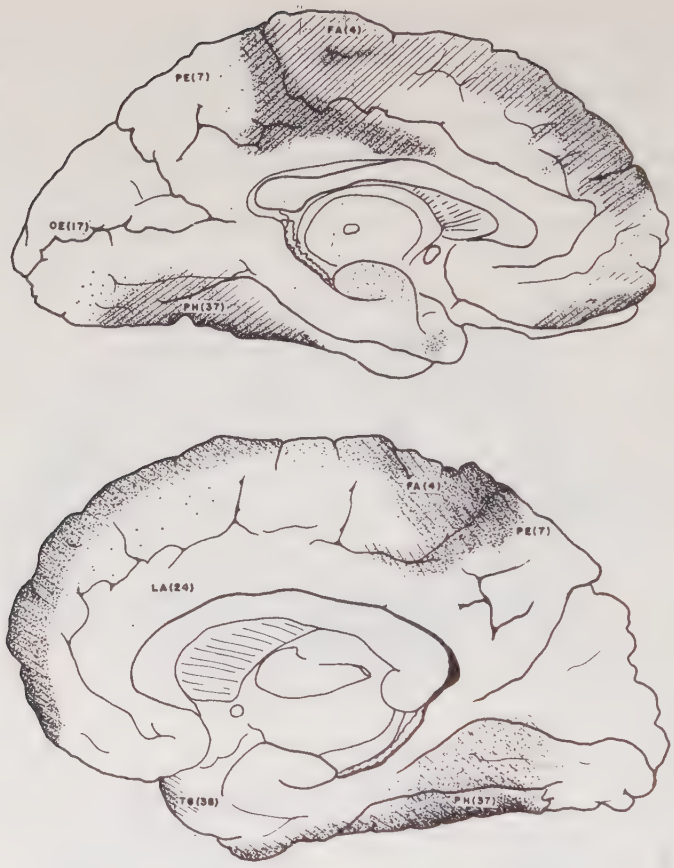

Fig. 9. Medial view of right and left hemispheres. Observe the preservation of limbic cortex, visual areas. Leg motor areas severely affected.

of carbon monoxide poisoning. The cerebellar lesions in this case were surprisingly mild.

The areas of cortical destruction (shaded areas with dots in the maps) correspond in general to the areas of subcortical myelin loss (oblique dashes). Only in the tips of the temporal lobes can one see a striking dissociation. One must assume that the cortex here was functionally inoperative, the intensity of subcortical fibre loss being such that both temporal tips were nearly devoid of connections.

IV. DISCUSSION

This patient was subjected to anoxia as a result of illuminating gas poisoning at the age of 21 and survived ten years. The findings in the brain were typical of hypoxic damage although some exceptions will be noted later. Since the patient suffered from hemodynamic shock during her period of hypoxia, the possibility that some of the lesions were ischemic must also be entertained. It should be kept in mind that similar patterns of language disturbance can be seen in patients as a result of great vessel insufficiency which can produce the same distribution of destruction of the brain.

Our patient showed the characteristic posture of cerebral paraplegia in flexion which has been thoroughly discussed by Yakovlev (1954) in his classic paper. Like his patients our case showed the characteristic lesions of the frontal lobes and the globus pallidus, while the medial lemnisci and superior cerebellar peduncles were intact.

The most striking clinical feature of this patient, and the main topic of this paper, was her language behaviour. Let us first comment on the eventual disappearance of this behaviour in her later years of hospitalization when she lost to a great extent her spontaneous utterances, her echoing and her learning of new verbal material. It seems likely that the presence of an ongoing pathological process can be excluded both by the history and the post-mortem findings. The presence of superimposed pathology of another variety to account for the change can also be excluded. Such deterioration of cerebral function in the face of fixed cerebral pathology is most commonly the result of superimposed medical illness, in particular infections and fevers. This patient had recurrent episodes of illness, e.g. recurrent bouts of pyelonephritis, and a hip fracture with subsequent amputation during her course. These were well-controlled, however, and her medical state was in general stable. She remained remarkably free of bed sores. The post-mortem also showed no evidence of severe medical illness, and the most positive finding was a focal healed pyelonephritis. One can clearly not rule out the possibility that medical illness accounted for her decline, but the question must remain open.

A third possibility is that progression of symptomatology occurred because of changes in the nervous system in response to a fixed lesion. Such changes are most familiar to neurologists in the natural history of spinal paraplegias or quadriplegias and may go on over long periods of

time. While this possibility must be considered in this case, it cannot be proven by the data at hand.

In an earlier report on this patient what was stressed was the apparent discrepancy between her absence of spontaneous language and her ability to sing and it was hoped that the pathology of this case might throw some light on the anatomical substrate of musical ability (Segarra and Quadfasel, 1961). On review of the case, however, it appears clear that this discrepancy was not so marked as it appeared at first. The patient could indeed sing correctly both words and music and learn the words and music to new songs. She could, however, repeat well-learned verbal material just as readily although more effort was devoted by her examiners to singing than to non-musical material. In fact, the discrepancy between musical and verbal ability mentioned is probably seen much more frequently in routine cases of aphasia, where it is common experience that patients who have little or no speech either spontaneously or in repetition, will correctly sing melodies without words. Our case does not aid materially in localizing musical abilities nor does it cast light on the discrepancies mentioned above which are observed in many aphasics. The explanation for these perhaps lies in the preservation of those regions in the right hemisphere which are homologous to the speech areas, but this can be resolved only by further studies of such cases.

Over several years the patient's speech behaviour remained essentially constant. The patient lacked all propositional speech and all evidence of comprehension except in certain specialized verbal responses. Her spontaneous speech consisted of only a few palilalic utterances. By contrast she showed echolalic repetition with excellent articulation. She manifested the 'completion' phenomenon characteristic of echolalia (Stengel, 1947). These phenomena taken together would clearly place this patient in the category of transcortical aphasia. The paucity and stereotypy of spontaneous speech and the total lack of comprehension, contrasting with the intact echolalic repetition would place her in Goldstein's category of mixed transcortical aphasia. As we commented in the introduction, Goldstein (1917) claimed that this syndrome would generally result from a characteristic pattern of lesions. There should be sparing of Wernicke's area (as well as the auditory pathways up to and including Heschl's gyrus), Broca's area (as well as the lower end of the Rolandic cortex and its motor outflow), and the connections between them. By contrast the lesions

should destroy the posterior (temporo-parietal) and the anterior (frontal) association cortex or should be so placed as to destroy the connections between Wernicke's area and the posterior region and between Broca's area and the anterior region.

Goldstein's thesis has the important implication that the brain of a patient with this syndrome will have cortical lesions which are extensive, but which will selectively spare certain structures. It is this selective pattern of sparing which accounts for the striking preservation of certain complex activities in the face of an otherwise dramatic loss of most other activities. It can be easily seen that the lesions found in our patient are strongly consistent with Goldstein's assertion. Broca's area (Figure 2) and Wernicke's area (Figure 3) were both intact. The lower end of the Rolandic cortex and Heschl's gyrus (Figure 3) are also intact, in addition to the medial geniculate body and the auditory radiation. The arcuate fasciculus extending from Wernicke's area to Broca's area was also intact (Figure 3). It would appear that this lesion accounts for the syndrome since Wernicke's area can no longer arouse associations elsewhere in the brain, except in Broca's area. The intact pathway from Wernicke's area to Broca's area accounts for the intact repetition. There is thus striking isolation of the intact speech area.

We feel that this anatomical arrangement also accounts for the 'completion' phenomenon (Stengel, 1936, 1947). The conformation of the lesions makes it impossible for auditory stimuli to arouse associations other than in auditory association cortex or in Broca's area. It is, however, perfectly reasonable to assume that purely auditory associations can be aroused. Hence, the first part of a conventional spoken phrase might quite easily arouse the remainder of that phrase. A less familiar or not previously known phrase has no other associations but can simply arouse direct repetition.

This explanation accounts not only for the 'completion' phenomenon but also for certain other characteristics sometimes seen in echolalic speech. Thus when an echolalic patient is told, "Say 'San Francisco'" he will frequently utter the words, "San Francisco" omitting the word "Say". It is easy to see that such differentiated responses can be learned entirely within the intact circuit including Wernicke's and Broca's areas and do not require connections to the remainder of the brain. Similarly some echolalic patients may echo when spoken to in their own language,

but not in a foreign language nor when nonsense words are spoken to them. The same explanation could quite readily apply to this phenomenon.

The other and perhaps most dramatic aspect of this patient was her ability to carry on verbal learning in the face of her extensive brain damage and her totally helpless state. She was able as we have noted to learn new songs. It appears to us likely that this ability to carry on learning was related to the relative preservation of the hippocampal regions on both sides (Figure 1). This was a somewhat surprising finding since anoxic damage to the brain typically is very destructive of the hippocampal formation. The rather good preservation of the hippocampi is further substantiated by the fact that the fornices appear to be roughly of normal size (Figure 4). It would seem reasonable that for verbal learning to take place, not only should Wernicke's area be intact but also at least the left hippocampal region and the connections between these two areas. The connections of the superior temporal gyrus to the hippocampal region are unfortunately not known in detail. A further study of these connections would be most useful to verify that they are indeed preserved in our case. Certainly the appearances of the sections of this case are compatible with the existence of a pathway between these regions but are certainly not adequate to prove this point. The preservation of significant verbal learning ability in the absence of significant propositional speech or comprehension has previously been observed in some children who are otherwise mentally defective, but has not to our knowledge been described previously as a result of acquired lesions in the adult. It would be most important to ascertain whether the brains of these children have a pathological pattern similar to that described here.

A distinction should be made between the extensive ability to repeat occurring in patients such as ours and the rather limited echolalia occasionally seen in some aphasic patients, particularly those with Broca's aphasia or conduction aphasia whose ability to repeat is severely limited. We believe that these latter patients echo within the limitations posed by their ability to repeat while patients such as ours may show a much more extensive ability to repeat consistent with the excellent preservation of the speech regions.

A final methodological point should be made. There is a general tendency to avoid clinico-pathological analysis of patients with multiple lesions. A closer attention to the correlation between what is preserved

clinically and what is spared pathologically may contribute further to our knowledge of the higher functions.

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NOTES

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CHAPTER XVII

HUMAN BRAIN: LEFT–RIGHT ASYMMETRIES IN TEMPORAL SPEECH REGION

PREFACE

At the time Walter Levitsky and I published this paper the prevailing view was that there were no significant anatomical asymmetries between the hemispheres and that the cause of cerebral dominance would have to be sought in purely physiological or in subtle anatomical differences between the two sides. Since the publication of this paper the results reported here have been replicated by several groups. Juhn Wada's report at the International Neurological Congress in 1969 not only confirmed our findings in adults, but also reported the presence of these asymmetries in the brains of fetuses and new-borns. More recently Teszner (1972) in Paris and Witelson and Pallie (1973) in Canada have added further confirmations. The statistics in all these series have been very close in value. Dr. William Sheremata and I have as yet unpublished data showing a correlation between the larger left temporal plane and the longer left occipital horn described by McRae *et al.* (1968). As a result of this correlation it will be possible to make a rough prediction as to the presence of anatomical asymmetry on the basis of pneumoencephalographic findings. The development of elaborate computerized scanning devices should make it possible to determine the size of the left occipital horn non-invasively. In fact it is possible that by suitable programming it will be possible to determine directly the size of the temporal plane on the two sides. Obviously the ability to determine in life by simple non-invasive techniques the presence and extent of anatomical asymmetries should have a major impact on studies of dominance and should thereby help to clarify the as-yet elusive nature of left-handedness, and possibly add to the understanding of delays in language and reading.

Lemay and Culebras (1972) have been able to demonstrate further anatomical asymmetries visible in arteriography. Furthermore they have pointed out the remarkable fact that one of these asymmetries, i.e., the tendency of the right Sylvian fissure to slope upward more sharply than

the left, is visible in the endocranial cast of the la Chapelle aux Saints skull of Neanderthal man dating back over 30 000 yr. This is the first solid piece of evidence as to the evolution of the changes in the brain responsible for language.

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HUMAN BRAIN: LEFT-RIGHT ASYMMETRIES IN TEMPORAL SPEECH REGION

1968

ABSTRACT. We have found marked anatomical asymmetries between the upper surfaces of the human right and left temporal lobes. The planum temporale (the area behind Heschl's gyrus) is larger on the left in 65 percent of brains; on the right it is larger in only 11 percent. The left planum is on the average one-third longer than the right planum. This area makes up part of the temporal speech cortex, whose importance is well established on the basis of both anatomical findings in aphasic patients and cortical stimulation at operation.

It is generally accepted that the preponderance of the human left hemisphere in speech functions is not associated with significant structural differences between the two halves of the brain (von Bonin, 1962). We reinvestigated this problem on an extensive sample and found highly significant differences between the left and right hemispheres in an area known to be of significance in language functions.

Our material consisted of 100 adult human brains, obtained at postmortem, and free of significant pathology.[1] The hemispheres were divided, and then the upper surface of the temporal lobe (supratemporal plane) was exposed on each side by a cut made in the plane of the Sylvian fissure. Figure 1, a drawing of a typical specimen, illustrates the anatomical landmarks. Figure 2 is a photograph of a specimen which demonstrates the typical left-right asymmetries. The posterior border of the planum temporale slopes backward more sharply on the left, while the anterior border of the planum (formed by the sulcus of Heschl) slopes forward more sharply on the left; both effects combine to produce a larger planum temporale on the left. A sharper backward slope was found on the left in 57 percent and on the right in 18 percent ($P<0.001$), with equality on the two sides in 25 percent of the brains examined. A sharper anterior slope was found on the left in 40 percent and on the right in 24 percent ($P<0.05$), with equality in 36 percent. The planum temporale was larger on the left in 65 percent and on the right in 11 percent ($P<0.001$), with equality in 24 percent of our specimens.

The length of the outer border of the planum temporale ($x-y$, Figure 1)

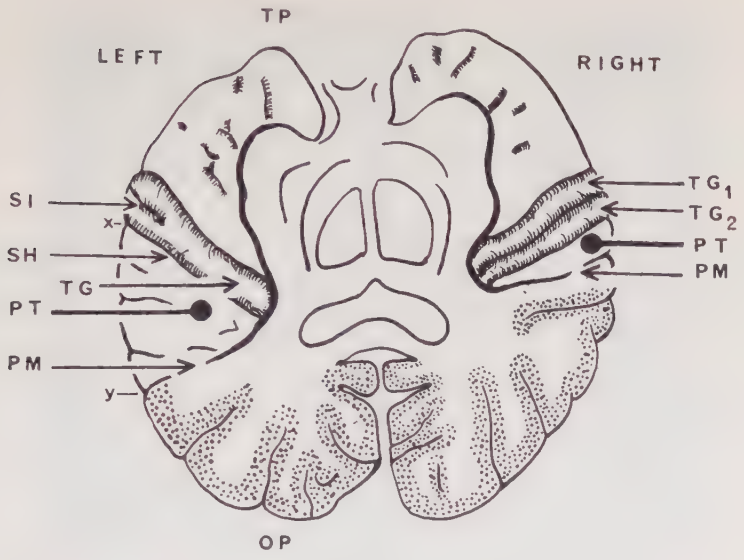

Fig. 1. Upper surfaces of human temporal lobes exposed by a cut on each side in the plane of the Sylvian fissure; anatomical landmarks and typical left-right differences are shown. The posterior margin (*PM*) of the planum temporale (*PT*) slopes backward more sharply on the left than on the right, so that end *y* of the left Sylvian fissure lies posterior to the corresponding point on the right. The anterior margin of the planum formed by the sulcus of Heschl (*SH*) slopes forward more sharply on the left. In this brain there is a single transverse gyrus of Heschl (*TG*) on the left, but two on the right (TG_1, TG_2). *TP*, Temporal pole; *OP*, occipital pole; *SI*, sulcus intermedius of Beck.

was 3.6 ± 1.0 cm on the left and 2.7 ± 1.2 cm on the right ($P < 0.001$); the planum was 0.9 cm or one-third longer on the left than on the right. These measurements are compatible with observations that the left Sylvian fissure in man is on the average longer than the right (von Bonin, 1962; Connolly, 1950). Our data show that this difference is accounted for by the increased length of the left planum temporale. Handedness data were, for reasons beyond our control, not available to us. Since, however, about 93 percent of the adult population are right-handed, while 96 percent are left-brained for speech, our 100 cases must have consisted overwhelmingly of subjects that were left-brain dominant for speech. It would of course be most useful to study the patterns found in the brains of right-hemisphere-dominant subjects.

These studies support earlier assertions, in studies lacking quantitative

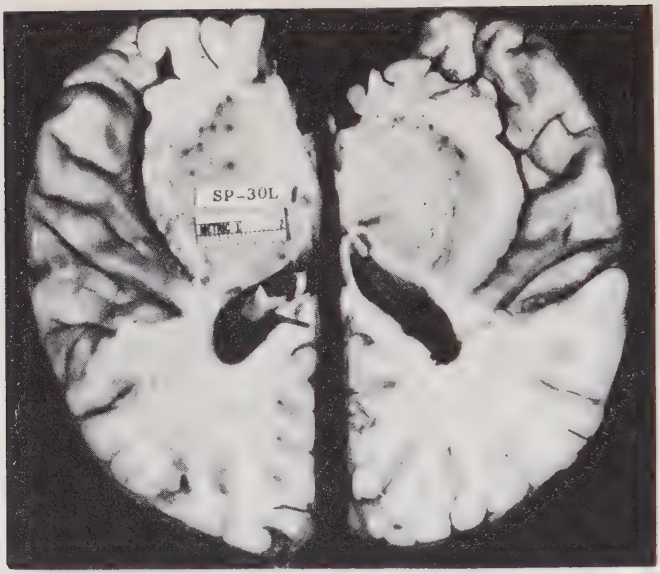

Fig. 2. Upper surfaces of temporal lobes exhibit typical right-left differences. Sharper backward slope of posterior margin and sharper forward slope of anterior margin of planum temporale on the left, larger planum on the left, and longer outer border of left planum are evident.

data or based on small samples, that the supratemporal plane showed marked right-left asymmetries in man (Flechsig, 1908; Pfeifer, 1936; von Economo and Horn, 1930). Pfeifer (1936) and von Economo and Horn (1930) found by contrast no asymmetries in the same region in anthropoid apes.

While Heschl's gyrus contains the primary auditory cortex (*TC*) the planum temporale contains auditory association cortex (areas *TB* and *TA*) which extends on to the lateral surface of the posterior portion of the first temporal gyrus (von Economo and Horn, 1930). These regions of auditory association cortex on the left constitute the classical Wernicke's area, a region known from anatomical findings in aphasic patients (Zangwill, 1960; Meyer, 1950) and from stimulation studies during neurosurgical procedures (Penfield and Roberts, 1959) to be of major importance in language functions. Our data show that this area is significantly larger on the left side, and the differences observed are easily of sufficient magnitude to be compatible with the known functional asymmetries.[2]

NOTES

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[2] Some of the work was carried out at the Boston Veterans Administration Hospital. Supported in part by grant NB-06209 from the U.S. Public Health Service.

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CHAPTER XVIII

DEVELOPMENTAL GERSTMANN SYNDROME

PREFACE

Frank Benson and I published this paper for several reasons. Kinsbourne and Warrington had already reported the presence of Gerstmann's syndrome in childhood. Our first case was of interest, however, since he was a child of superior intellectual capacities without any history of dyslexia.

This case illustrates well the problem of using a single measure of 'intelligence'. He could read extremely well and carried on a high level conversation on such topics as politics but had persistent difficulty in mathematics and right-left orientation. Similarly some children with severe developmental reading difficulty may show strikingly good ability in drawing. All these suggest that one must presume the existence of special talents. Naturally the argument persist as to whether there is also not some 'general' factor, but the precise nature of this factor has never been adequately specified. I tend to be skeptical, however, that there is any one talent that could be given the name 'general intelligence'. Obviously I cannot in this introduction present a discussion worthy of this old and vast topic. I would suggest, however, that perhaps the study of the brain and its disorders may be a useful way of attacking it.

DEVELOPMENTAL GERSTMANN SYNDROME*

1969

ABSTRACT. We have presented 2 cases with an apparent developmental mental syndrome. We call this Gerstmann syndrome because of the presence of the classical tetrad of right-left disorientation, finger agnosia, agraphia, and acalculia. Reading ability was strikingly preserved in both cases. That the findings in these cases actually represent some underlying disorder (i.e., disordered spatial orientation, defective body image, or visuomotor apraxia) is quite possible, but until additional cases have been investigated and fuller explanations are available, we suggest they simply be considered examples of a developmental Gerstmann syndrome.

During the past decade increasing interest has been given to the existence of specific learning disorders in children. Childhood dyslexia has been the most studied and discussed of these syndromes but other varieties of learning disorder have been suggested. This paper will present a well-defined developmental syndrome which can be differentiated from other learning disorders of early life. We will stress, in particular, the absence of dyslexia in this syndrome.

I. CASE 1

A 12-year-old boy was referred to the Aphasia Research Center of the Boston University School of Medicine because of threatened school failure. In particular, significant spelling disability jeopardized continued school promotion. He was the oldest child in a family of 4, the 3 younger siblings having no recognized learning difficulty. Both parents were well educated, and the father was a highly successful professional man. Birth and developmental history were entirely without abnormality. He was fully right handed although the father was left handed. The patient had attended and performed adequately in both private and public schools; his achievements, however, were considered poor when compared with those of his brother, who was one year younger and rated 'near genius' by psychological testing. Spelling difficulty was first recognized in third grade and has persisted despite family and school efforts toward improvement. In striking contrast to the disturbance in spelling, reading ability has

always been superior. In a test given in an excellent public school system, he was found to read 360 words per minute with 82% comprehension and thus ranked as the best reader in the school system for his age group. At the time of evaluation his reading activities were precocious. He had just finished reading *The Rise and Fall of The Third Reich* and was reading *Silas Marner*. He had read nearly all of the works of Poe, his favorite author. His hobby was stamp collecting but he enjoyed many participation sports including tennis, basketball, swimming, and football. Personal and social adjustments were excellent. Past history included no significant disorders except for two fractures of the right leg, one occurring in a fall down stairs and the other as the result of a skiing accident. In neither accident had there been any significant head injury.

A full neurologic evaluation revealed no abnormality. Coordination was quite normal for age. Muscle tone, reflexes, and sensation were all within normal limits. Eye movements were intact, as were the remainder of the cranial nerves. Testing of recent and remote memory revealed no abnormality. He showed considerable insight, personal interest, and concern, appropriate to the testing situation. Cooperation was excellent.

Spontaneous speech was fluent and well articulated, had good melody, and revealed an exceptionally fine vocabulary. Comprehension of spoken language was tested thoroughly and was normal in all spheres. Similarly, repetition of spoken language was entirely normal. There was no demonstrable defect in word-finding ability. He was able to present lengthy lists of words on suggested topics. During sixty-second periods he named 20 animals, 15 makes of automobiles, and 19 words beginning with the letter B, all superior performances.

In reading aloud, his delivery was rapid and smooth, with only occasional omission of a word, most often a grammatical filler, and no paraphasic substitutions. Comprehension of written language was tested using paragraphs followed with either true-false or multiple choice questions. Some paragraphs were from the *Reader's Digest* and others from a reading aptitude test at high school senior-college freshman level. He had no difficulty with any of this material. In contrast to this ease of reading, there was notable difficulty in writing. His script was fairly legible, with small, clumsy letters. When asked to write sentences describing the weather, the profession of his father, or his route to school, the sentences were simple in structure (almost always subject-verb-object)

and the vocabulary was elementary, a striking contrast to his spoken vocabulary. When asked to spell specific words, the writing problem became more manifest. He spelled druggist as 'drugest' hospitable as 'hospitible', yacht as 'yought', and telephone as 'telaphone'.

He could draw a circle, square, and triangle on command, but when he was asked to draw an animal, he merely drew a wavy line with a small circle at one end and called this a snake. He was unable to show the third dimension when asked to draw a box or a cube. His attempts to copy drawings were crude and also failed to demonstrate the third dimension. He correctly performed the mannequin puzzle but this took forty seconds of concentrated effort. He had great difficulty with Koh's block designs, totally failing elementary four-block designs. If a model was made by the examiner and left in view, he copied it successfully but showed 'closing-in phenomena' (i.e., the tendency to bring his copy closer and closer to the model). He was able to differentiate right and left on himself after considerable latency and after cuing himself by moving his right hand. If the examiner sat at 90° from the patient and held up one hand, the patient performed randomly in naming the requested hand. He usually chose correctly if the examiner was directly opposite him but made occasional errors and always showed a long latency. He could name only three fingers but could point to the finger named by the examiner. A simple finger-localization test was given. With one hand behind his back and the other on the table in front of him, one of the fingers on the hidden hand was stimulated by the examiner. The patient consistently failed to move the same finger of the exposed hand as requested. Calculations were done poorly and only at the simple rote-memory level. There was difficulty in column placement in both addition and multiplication problems. All calculations, even those in which he produced a correct answer, were done with effort, hesitancy, and uncertainty.

A full Wechsler Adult Intelligence Scale test was performed, showing a verbal score of 131 and a performance score of 101. The verbal score was uneven and depressed by a very low score in arithmetic, a pattern which suggests an even higher level of accomplishment in purely verbal activities. The family and the school authorities were informed of our findings and suggestions were made to both concerning management of his education. In particular, the school was encouraged to accept oral

reports and oral test procedures. When last seen, the patient had not only attained appropriate promotion but was ranked in the upper one-third of his class.

In summary, a 12-year-old boy, referred because of threatened school failure, showed superior oral language function and superior comprehension of written material. He manifested distinct abnormalities in writing, calculating, differentiating right from left, localizing his fingers in space, and constructional tasks. The very high level of performance in other cortical functions contrasted sharply with these five failings, the first four of which made up the Gerstmann syndrome. There was neither history nor clinical evidence of brain injury as a source of this syndrome, suggesting, therefore, a probable developmental etiology.

II. CASE 2

A 13-year-old white girl was referred to the Aphasia Research Center after examination by two neurologists. These examinations had been suggested by her school because of difficulty in mathematics, contrasting with adequate performance in other subjects. At the time of examination, she was in seventh grade and passing all courses except mathematics.

The patient was a product of artificial insemination and delivered under careful obstetrical care with no complications. Birth weight was 6lb. 3oz. The mother noted considerable weight gain during pregnancy and there was some question of a toxemic state at the time of delivery. The patient remained in an incubator for two days and the mother states that the patient had 'blue feet'. The developmental milestones were appropriate; she walked at 15 months and is said to have talked at an early age.

Family history concerning the father is unobtainable. The mother admits a lifelong difficulty with mathematics but has been able to work as a cashier in a restaurant. The patient has always preferred the right hand for unilateral activities and there is no family history of left handedness. While the patient has always been considered awkward, she learned to ride a bicycle and to roller skate proficiently at an appropriate age.

Past history was negative for significant head injuries, major surgery, or serious illnesses. There had been no headaches, convulsions, or other neurological symptomatology. She was examined at an early age for thyroid deficiency but no specific abnormality was demonstrated. She

spent one year in a special school because of 'behavior difficulties'. She was asked to leave because she was "so much better than the other children that she was out of place in that setting." Her school record was satisfactory except for the difficulty in arithmetic. She had been receiving high marks in reading up to the time of the present examination.

Careful neurological examination was performed by three neurologists and no definite abnormalities were observed. Several 'soft signs' were noted, however, the most pertinent were slowness and awkwardness in performing rapid alternating movements, present bilaterally but more pronounced on the right, some spooning of the extended right hand, and a slight increase in passive tone in the right extremities. There were no abnormalities in reflexes, sensation, gait, vision, or extraocular movement. Mental status evaluation, exclusive of language, suggested that the patient's fund of knowledge was limited, consisting mostly of material associated with her school activities. Formal memory testing demonstrated an adequate ability to learn new material. She intimated a lack of interest in the examination but with actual testing she became cooperative and at no time did the examiner feel that she performed indifferently.

Her spontaneous speech was, at first, sparse but with time she relaxed and finally spoke quite freely. In testing of serial speech she counted and recited the days of the week without difficulty but had trouble reciting the months of the year. She sang with good melody and words. Comprehension tests, including yes-no questions and requests to point to objects in the room and to perform actions, were all carried out well. She could repeat without difficulty. A digit span of five forward was consistently demonstrated. She could name most objects on confrontation but often had difficulty in naming parts of the objects. Evidently she had not known many of the names of the object parts prior to evaluation. Her performance in naming body parts and colors was below anticipated level. She was, however, able to present a list of 19 animal names in a period of sixty seconds.

She read aloud without difficulty, and comprehension of written material was adequate for her age. While she had difficulty understanding written material at the high-school level, she comprehended adequately below this level. Writing, on the other hand, was grossly abnormal, with large, clumsy script. When asked to write a sentence describing what she was going to do later in the day, she made one grammatical error and

badly misspelled one of the words. When words were dictated, she made many spelling errors, some of which were phonetic while others consisted of omissions or reversals of letters.

Right-left orientation was difficult for the patient. She was usually correct on her own body but only after considerable hesitation. She performed randomly when the examiner sat opposite her and asked her to point to his right or left side. Finger identification was also done poorly. She could name only one of the fingers and point to only two of the fingers when the names were given, a marked contrast to her ability to name other body parts. She failed totally at finger-localization tests. Her competence in calculation was extremely low. She was able to write single digits to dictation but when asked to write numbers in the hundreds or thousands, added extra zeros so that 451 was written as 40041. She correctly performed simple addition problems only by counting on her fingers. More complex addition problems were not worked and she was never able to perform subtraction problems. Marked difficulty was seen in construction tests. She put the mannequin puzzle together incorrectly in thirty seconds and was satisfied with the result. She could not perform any of the Koh's block designs unless a model was made for her to copy.

Psychological testing (WAIS) demonstrated a low IQ. The verbal IQ was registered at 79 with the performance IQ at 75. These scores were depressed by the subtests involving arithmetic and construction. It was considered that she could be placed in the dull-normal range if these specially affected areas were omitted.

Following this examination, contact was made with the school authorities and arrangements were made to obviate some of the more significant difficulties. On this basis, she has maintained status in the appropriate class, is doing work satisfactory to the school system and, apparently, will continue education through high school.

In summary, this patient had a learning problem which was noted in the school as a specific difficulty in mathematics. Both clinical impression and psychological testing show a generally dull girl. Evaluation of cortical function revealed the components of the Gerstmann syndrome (right-left disorientation, finger agnosia, dysgraphia, and severe calculation disturbance) plus constructional apraxia. These disabilities apparently accounted for most of her scholastic difficulties. In contrast, oral language and ability to read were at a much higher level. The lack of pertinent

neurologic history suggests that this is a developmental defect, although evidence for this is not as strong as in Case 1. Corrective measures within the school have allowed her to continue in an adequate educational program.

III. DISCUSSION

The above cases had 5 striking abnormalities: dyscalculia, dysgraphia, right-left disorientation, finger agnosia, and constructional apraxia, of which the first four make up the well-known Gerstmann syndrome. In a series of papers, Gerstmann (1924, 1927, 1931) described patients with this clinical combination, noted consistent dominant parietal pathology, and theorized that this symptom combination resulted from a partial autotopagnosia, a defect in recognition of one's own body. Other neurologists soon described cases with similar disorders, confirmed the predominant site of abnormality, but disagreed on the theoretical considerations. Thus, one group suggested apraxia involving the fingers as the basic deficit (Hermann and Pötzl, 1926); others suggested a disturbance of spatial orientation, particularly orientation of the body in external space (Lange, 1930, Stengel, 1944). All agreed on the occurrence of the syndrome and the consistency of dominant parietal pathology, and the Gerstmann syndrome because an accepted tool of neurologic localization.

More recently the existence of the syndrome has been questioned. Heimburger *et al.* (1964) showed that a single symptom from the tetrad could be seen in cases with small lesions located anywhere. All four findings were present only in cases with extensive cerebral damage, invariably accompanied by other symptoms and involving the dominant parietal cortex in about 90%. Benton and co-workers conducted careful studies over many years (Benton, 1959, 1961). They showed that the individual components were usually accompanied by other functional abnormalities and failed to demonstrate a high correlation between the four components. Benton concluded that the Gerstmann syndrome was an artifact of biased testing and represented only a chance grouping of symptoms among many produced by a large lesion. In a smaller study, Poeck and Orgass (1966) reached similar conclusions and found a high frequency of dysphasia in their cases, suggesting that the Gerstmann tetrad was a manifestation of an actual or latent aphasic condition. Despite the doubts cast on the interpretation of the syndrome, it remains clear that a domi-

nant parietal lesion is probable when all four components are present.

While the interpretation of the adult form of this syndrome may be controversial, the syndrome is not rare. In contrast, there are very few reports of the Gerstmann syndrome in children. Critchley (1953) credits a case described by Spillane (1942) as the original description of the Gerstmann findings on a developmental basis. Hermann and Norrie (1958), in a study of dyslexic (word-blind) children, noted an increased frequency of difficulty in right-left orientation and finger discrimination but did not mention the other components. They suggested that the dyslexia was developmental and that the Gerstmann syndrome components were part of the same underlying abnormality and concluded that dyslexia was a congenital manifestation of the Gerstmann syndrome. Kinsbourne and Warrington (1963) reported 7 children who demonstrated 3 or more of the Gerstmann components, most often associated with other neurological disability. All demonstrated a characteristic pattern on the WAIS, with the verbal score being considerably higher (from 20 to 35 points) than the performance score. The authors considered this a developmental variant of the Gerstmann syndrome which could be either constitutional or posttraumatic, could exist alone or with other neurologic symptomatology, and could cause reading and writing retardation. In a later review, Kinsbourne (1968) described this condition as a developmental cognitive deficit which could result from either localized cerebral dysfunction or maturational lag. He again noted reading retardation as a possible accompanying symptom.

Kinsbourne and Warrington noted, however, that serious dyslexia could occur without Gerstmann symptomatology and, conversely, normal reading ability is a possibility in some patients with the full Gerstmann tetrad (one of their cases was an exceptional reader). They concluded that, while the two abnormalities might coexist and influence each other, they should be considered separate disturbances.

The patients presented in this report have all four components of the Gerstmann syndrome plus constructional apraxia. Many authors have noted that constructional apraxia commonly accompanies the Gerstmann syndrome and could be considered an additional component.

We believe that all of the symptons present in the two patients reported here can be attributed to these five abnormalities. Case 1 demonstrates this most distinctly – an otherwise highly intelligent boy with considerable

disability in writing, calculation, and constructional tasks and demonstrable abnormalities in finger discrimination and right-left orientation. To the best of our knowledge there is no previous report of developmental dysfunction showing the components of the Gerstmann tetrad in an individual of such superior verbal intelligence and, in particular, superior reading ability. This contrast dramatically highlights the specific disabilities producing the depressed Wechsler performance score and serious scholastic difficulties. The second case is less clear-cut, but a review of the findings demonstrates that the Gerstmann syndrome deficits are significantly more severe than her other shortcomings. It seems likely that Case 2, with apparent intellectual deficiency overshadowing the specific deficits, may be afflicted with a fairly common variety of learning disorder.

These cases clearly demonstrate that the Gerstmann syndrome variety of learning disability may exist quite independently from dyslexia. Case 1 was an exceptional reader, well above grade level despite serious limitations in both writing and calculation. Even Case 2 had much greater success in reading than in either writing or arithmetic. Thus, in these two cases of developmental Gerstmann syndrome, dyslexia is notably absent. Nonetheless, these children have a disorder which can be classed with the specific learning disorders, both subjects performing far below grade level in selected activities. We would suggest that the developmental Gerstmann syndrome represents a specific learning difficulty which can be easily differentiated from dyslexia. We would stress, also, that severe difficulties in writing and spelling can occur without difficulty in reading. Such cases may be common and erroneously called dyslexic because of the difficulties in spelling and writing.

Very little can be stated concerning the pathology underlying these two cases. Both children are healthy and have no other obvious neurologic abnormality or any history suggesting perinatal or postnatal cerebral disease. In this way, they differ from the cases reported by Kinsbourne and Warrington (1963), of whom 5 had obviously sustained perinatal trauma, 1 had suffered severe septicemia as an infant, and 1 showed considerable accompanying neurologic and behavioral abnormality. The present cases appear to be examples of developmental abnormality, but whether this represents a maturational lag, as suggested by Kinsbourne (1968), can only be answered by additional follow-up. The accepted pathology underlying Gerstmann syndrome in the adult cannot necessarily

be attributed to developmental cases. In a recent report, Money (1964) noted a frequent difference in verbal and performance subscores in patients with Turner's syndrome (chromosome count 45, XO, female characteristics). In a significant majority, he noted a higher verbal subscore (median, 104.6) than performance (median, 87.6). Unfortunately, the author makes no reference to components of the Gerstmann syndrome or to reading ability in this group. Neither of our patients has this specific genetic abnormality, but the possibility of the existence of some other genetic abnormality can be considered.

Clinically, the developmental Gerstmann syndrome is fairly distinct. Appropriate testing of reading, writing, arithmetic, and constructional skills and attention to right-left orientation and finger discrimination offer a clear picture. A strong difference in WAIS scores, with the verbal score highest, has been seen in most of these cases. Probably there are many cases similar to the two described here which are presently mislabeled as either dyslexia or mental retardation; appropriate testing could place them in the correct diagnostic category.

The desirability of making this diagnosis is apparent from the results in these two cases. Minimal environmental manipulation at home and in school produced beneficial results in both cases. Case 1 rose to the upper one-third of his class after he was allowed to give oral rather than written reports and his course schedule was shifted to take advantage of his superior reading ability. Case 2 has not done as well, but similar school manipulation has allowed her to remain with her class and there is promise of accomplishing high school graduation. In both situations, informing the school officials of scholastic strengths as well as defects allowed alteration of educational activities to offer maximum opportunity for learning. Just as significant was the psychological benefit obtained by outlining the defect to the families. In both cases, the presence of a specific learning disorder was acceptable and the ensuing support offered to the children was distinctly beneficial.

NOTES

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CHAPTER XIX

THE ALEXIAS

1969

Alexia may be defined as an incapacity in the comprehension of written or printed words produced by a cerebral lesion. In current usage alexia refers only to acquired defects in contrast to dyslexia, a term designating an innate or constitutional inability to learn to read (Critchley, 1967). This distinction has not gained universal acceptance but has become increasingly popular (Leischner, 1957) and will be employed in the following discussion. The classical term 'word blindness' is presently used by only a few investigators.

Many other terms have been employed to qualify the general term alexia. Most of these are best described in context with the particular symptom complex they denote but several are used to extensively that they deserve mention here. Literal alexia indicates an inability to recognize letters while verbal alexia denotes a state in which letters are recognized but words are not. This division has been employed in many classifications such as those of Hinshelwood (1900), Brain (1961) and Hécaen (1967). The question as to whether these are truly separate entities or points on a continuum will be discussed later.

'Wortblindheit' (attributed to Kussmaul, 1877) was the first widely used term describing acquired reading disturbances ('cécité verbale', commonly used by French authors and word blindness in English are exact translations). By analogy, various authors have spoken of letter blindness, mind blindness, 'Seelenblindheit', 'Notenblindheit' and 'Zahlenblindheit'. These terms are not frequently used at present and the term word blindness has been replaced almost completely by the more general term alexia.

Early investigators stressed the presence or absence of agraphia in cases of alexia. Alexia without either agraphia or significant aphasia has often been called 'pure' alexia ('reine Wortblindheit'). Several authors have asserted that there is no reported case of alexia without some other neurologic or neuropsychologic accompaniment (Beringer and Stein, 1930; De Massary, 1932) and question the use of the adjective 'pure' in

this disorder. For most authors, however, the word 'pure' does not imply an absolutely isolated alexia but stresses that alexia dominates the clinical picture. The term 'pure alexia' is thus still in use (Symonds, 1953; Gloning *et al.*, 1955) to describe alexia without significant agraphia or aphasia.

Many other terms have been used to differentiate varieties of alexia. Some are based on cortical localization (e.g. occipital or parietal alexia and even temporal and frontal alexia). Agnosia as an explanation of alexia has led to terms such as agnosic alexia, symbolic agnosia and even to such combinations as subcortical visual verbal agnosia or occipital agnosic verbal alexia (Nielsen, 1946). Goldstein (1948) suggested the more basic terms, primary and secondary alexia, to emphasize the absence or presence of associated aphasic disturbances. Some authors use total and partial as descriptive of the severity of alexia.

A difficulty in the assessment of the literature is the failure of some authors to differentiate the ability to read out loud from the comprehension of written material. By the definition offered in the first paragraph only failures of comprehension of written material may be designated by the term alexia. Those syndromes which may be confused with true alexia will be discussed in a later section of this chapter.

I. HISTORY

Benton (1964), in his review of aphasia before the time of Broca, cites four early reports of alexia. The oldest is credited to Valerius Maximus, who, in 30 A.D. described a man who, after being struck on the head by an axe, lost his memory for letters but had no other defects. Mercuriale in 1588 in describing a patient who had suffered a major seizure, noted "a truly astonishing thing; this man could write but could not read what he had written." In 1673 Johann Schmidt described two individuals with alexia, one who remained unchanged while the other responded to retraining. The last author quoted by Benton is Johann A. P. Gesner, who in 1770 described a patient with impaired reading who had more difficulty with his native German than he did with Latin. Genarin in 1838 and Trousseau in 1865 are credited by Charcot (1877) with early reports of reading disturbance in the French literature.

Despite the unique quality of these cases there was little interest in

alexia until the time of Broca and the subsequent interest in the neuropathological basis of language disturbance. Even then cases of alexia were infrequently recorded, at least partially because illiteracy was still widely prevalent. Broadbent (1872) recorded a case of severe reading disability in an individual with only mild aphasic disturbances and minimal disturbance of writing. He also recorded the first postmortem findings but, since the patient had succumbed to a large intracerebral hematoma involving the left hemisphere, definitive study was impossible. Broadbent did, however, note evidence of an old infarction deep in the white matter of the parietal-occipital junction and suggested that this area was probably important for the association of visual and verbal material. Kussmaul (1877) described this same symptom complex and is credited with the first description of a right homonymous hemianopia in conjunction with alexia. Guéneau de Mussy (1879) described a patient able to read numbers, but not letters or words. Bertholle's (1881) patient suffered from asyllabia, an ability to read letters individually but not in combination. This patient read only a few words in isolation, but by spelling out each letter in the word was able to read a phrase or short sentence. Charcot (1877) reported a patient with a right visual-field defect who could read only by tracing the letter with his finger. This patient could read individual and grouped numbers but had a distinct calculation disturbance.

The study of alexia was advanced considerably by Dejerine (1891, 1892) with the publication of two clearly defined case reports including postmortem findings. The first report (1891) described a 63-year-old patient, who, eight months before death, suddenly discovered that he was no longer able to read. Examination revealed only minimal right-sided weakness and a 'probable' right hemianopic visual-field defect. Verbal paraphasia was present in spontaneous speech and repetition but there was no difficulty with object naming. The alexia was total as was the agraphia except for the retained ability of the patient to write his own name. One month after onset the paraphasia in speech had disappeared but there was no improvement in reading or writing. When examined shortly before death the patient showed only slight change in the alexia (recognition of his own name and a few numbers) and no improvement in writing. The major finding at postmortem examination was an old, yellow infarct involving three quarters of the cortex of the angular gyrus and extending to the occipital horn of the lateral ventricle.

In the second paper Dejerine (1892) described an educated businessman who acutely lost the ability to read but had no evidence of aphasia, no disturbance of writing (spontaneously or to dictation), no difficulty in naming objects on visual confrontation and no difficulty in number reading. There was a right homonymous hemianopia (he was originally seen by the ophthalmologist Landolt who published his findings in this and other cases, 1888) but no loss of ability to name colors. The patient was followed until a second vascular accident produced total agraphia in addition to the alexia, still without significant aphasic symptoms. Ten days later the patient expired and at autopsy two separate infarcts were demonstrated. One was a softening involving a major portion of the left angular gyrus which by its pathological characteristics was of recent origin. The second was a yellowed, scarred lesion affecting the medial and inferior aspects of the left occipital lobe with additional involvement of the splenium of the corpus callosum. This older lesion lay entirely in the area supplied by the left posterior cerebral artery. Dejerine concluded that the occipital infarct had destroyed the visual pathways of the left calcarine area and the callosal lesion had severed the connections between the right hemisphere visual areas and the left hemisphere thus isolating the left parietal cortex from its usual routes of visual stimulation. He further conjectured that the dominant angular gyrus acted as a center for the optic images necessary for written language; if this area remained intact but isolated from the visual regions the patient retained writing skills despite severe alexia but if the angular gyrus was destroyed both reading and writing would be lost. The agraphia occurring shortly before death in this case was considered a result of the recent angular gyrus lesion.

Bastian (1898) supported the findings of Dejerine with several case reports and by 1900 Hinshelwood reported a series of 28 cases of 'word blindness' taken from the literature and including 6 from his own practice. Thus by the beginning of the 20th century the presence of reading disturbance, both as an isolated phenomenon and in relationship with agraphia or aphasia was well recognized and documented. In the ensuing years many cases have been added to the literature, most often in single case reports without pathological data. A myriad of neighborhood signs differing in many instances from case to case have been presented and quite often the prominence of one of these signs has led the investiga-

tor to suggest it as the primary cause of reading disturbance itself. Thus the 20th-century literature on alexia contains many suggested theoretical mechanisms for the production of alexia.

Several comprehensive reviews of alexia have appeared since the monograph of Hinshelwood. De Massary (1932) thoroughly reviewed the French contributions to the subject but was somewhat incomplete in reports of German and English articles. Lange (1936) reviewed the German contributions. Weisenburg and McBride (1935) and Holmes (1950) have reviews in English, again suffering from inadequate reporting of foreign contributions. More recently Leischner (1957) has published a monograph on disorders of written speech which includes a good review of alexia.

Disagreements as to the underlying mechanisms of alexia have been less marked in the eastern European than in the western literature. Many good reports concerning reading disorder have emanated from the eastern European countries, almost all of which reflect the powerful influence of pavlovian thinking. Thus alexia is discussed in terms of a defect in the primary or secondary visual analyzer (Davidenkov, 1956; Luria, 1966), the former referring to integrative activities in the region of the primary visual cortex while the latter indicates a separate area involved in complex visual associations. The recent work of Konorski (1967) in Poland has stressed the anatomical connections of these areas.

There are only a few reports from the Orient discussing alexia but these are significant for their comparison of phonetic script and the 'Chinese' character (ideographic) type of writing. Imura (quoted by Panse and Shimoyama), Asayama (1914) and Panse and Shimoyama (1955) discuss reading disturbance in Japan where both types of writing are used and understood by most of the population. Asayama described in detail a case of alexia produced by a dominant hemisphere luetic vascular lesion that produced almost total alexia and agraphia in Kana script (a phonetic syllabic script) while there was a negligible loss of ability to handle the 'Chinese' type script. This finding is supported by the other authors who note this dichotomy as the prevalent form of reading disturbance among the Japanese. They conjecture that the Kana script depends on auditory-visual (phonetic) function, more susceptible to damage in cases of aphasia than the more strongly visual 'Chinese' script. A single case (Lyman et al., 1938) of an educated Chinese financier, fluent in both Chinese and English,

with a surgically treated left parietal-occipital tumor indirectly supports this view by manifesting the opposite dissociation. Their case suffered severe disability in both reading and writing of Chinese characters while suffering only minimal disturbance with English written material. Their case had practically no aphasic residual and the authors conjecture that the posterior location of the lesion produced most severe interference with the more visually oriented Chinese character script. Further studies of alexia in oriental languages would certainly be useful for both anatomical and functional theorizing.

II. CLASSIFICATIONS

The presence of variations in the symptom picture of alexia was noted even in early observations. These variations involved both the reading disorder itself and the associated neurologic and psychologic signs. These differences led to classifications based upon individual opinion as to the importance of the various findings. Leischner's (1957) monograph includes a good survey of such classifications taken from the world literature and has been used as the basis for the following presentation. (An asterisk marks the classifications taken directly from Leischner's review.)

Wernicke* proposed the following three varieties of alexia:

(1) Cortical alexia – in which both reading and writing are severely disturbed.

(2) Subcortical alexia – in which reading is impossible but writing is preserved. These patients have a right homonymous hemianopia.

(3) Transcortical alexia – which produces errors in both reading and writing while copying is preserved.

In addition, Wernicke* suggested the following separation of alexic syndromes:

(1) Literal alexia – in which the patient finds it impossible to name even a single letter and rarely comprehends a letter.

(2) Verbal alexia – in which the patient can read and recognize single letters but has great difficulty in reading words.

Weissenberg* proposed the following classification:

(1) Alexia – a loss of the ability to read letters which could be total or partial (hesitant reading with many errors).

(2) Dyslexia – in which letters are read aloud and apparently understood but can not be combined for the comprehension of words. This too can be total or partial.

It has already been noted that the term dyslexia is no longer popularly used in this manner and most authors have preferred the terms literal and and verbal alexia for the entities mentioned by Weissenberg.

Dejerine* in a series of presentations carefully described several varieties of alexia and introduced the following classification:

(1) Word blindness and agraphia ('cécité verbale avec agraphie'). The patient can neither read nor write, even with help of somesthetic kinesthetic aids.

(2) Pure word blindness with preservation of spontaneous and dictated writing ('cécité verbale pure avec intégrité de l'écriture spontanée et sous dictée'). The patient cannot read, but writes to dictation or spontaneously. He cannot, however, read what he has written.

Pick* introduced a new concept in the following classification:

(1) 'Störungen der Wortformfassung' (word-form blindness, defect of recognizing the appearance of a word). This occurs when the patient can no longer distinguish the form of a letter. The patient can neither read aloud nor understand when reading silently.

(2) 'Störungen der Wortsinnerfassung' (word-meaning blindness, defect of comprehension of the meaning of a word). Here the patient can read words and letters aloud, but fails to recognize the meaning of what he has read.

Goldstein* simply divided alexia into primary and secondary varieties:

(1) Primary alexia – this is a comparatively isolated disturbance of reading which the author considered to be either a variety of visual agnosia or a defect in the ability to handle abstract language material.

(2) Secondary alexia – by this was meant that the alexia was part of a more complex language disturbance with major aphasic difficulties.

Hermann and Pötzl* proposed a classification based on Dejerine's:

(1) Pure word blindness without agraphia.

(2) Alexia-agraphia of the parietal type.

Kleist* suggested three types of alexia which in essence conform to Dejerine's classification:

(1) Subcortical alexia.

(2) Corticocommissural alexia.

(3) Cortical alexia.

The first two varieties have right visual-field defect and produce alexia by separation of the intact right visual cortex from the portion of the left hemisphere necessary for understanding written material while the third type develops after damage to this left cortical area itself.

Misch and Frankl* emphasized in their classification the presence or absence of visual agnosia:

(1) Alexia accompanying a severe visual agnosia – a state in which recognition of words was lost along with all other visual recognition.

(2) Alexia as the primary symptom of an otherwise mild visual agnosia.

(3) Alexia without recognizable visual agnosia. These patients can read, but only with difficulty.

Joy* also divided reading disturbances into three varieties but utilized clinical and psychological terminology:

(1) Aphasic alexia – in which the ability to understand words is lost.

(2) Semantic alexia – in which simple words are recognized, but not more complex words.

(3) Agnosic alexia – in which the graphic symbols are not recognized (visual agnosia).

Nielsen* also used aphasic, agnosic and semantic divisions in classifying alexia but included anatomical localization in his classification:

(1) Agnosic alexia – in which the lesion is in the dominant occipital white matter, often accompanied by a lesion in the splenium of the corpus callosum.

(2) Aphasic alexia – in which the lesion involves the angular gyrus of the dominant hemisphere.

(3) Semantic alexia – in which the lesion involves Wernicke's area of the dominant posterior temporal cortex.

Luria (1966) divided alexia into two main groups:

(1) Visual alexia – in which he defines two further subclasses, literal and verbal alexia, the former considered a variety of visual agnosia while the latter is a variant of simultanagnosia.

(2) Reading disturbances secondary to aphasic disturbances.

Brain (1961) returned to the familiar varieties of Dejerine, although with new terminology:

(1) Agnosic alexia – a failure to recognize visual symbols of speech with retention of the ability to write.

(2) Visual asymbolia – the inability to read is accompanied by difficulty in writing.

Hécaen (1967) has proposed the following classification of alexia:

(1) Aphasic alexia – the reading disturbance that accompanies Wernicke's aphasia.

(2) Alexia with agraphia – the combination of severe reading and writing disturbance in a patient with little evidence of aphasia.

(3) Alexia without agraphia – the presence of significant reading disturbance without aphasia or defect in writing. This syndrome is further subdivided into three subgroups: (a) literal alexia, (b) verbal alexia, and (c) global alexia.

Despite variation in the terminology employed most of these classifications are remarkably similar indicating the existence of clinically distinct entities. We will use the following, simple clinical classification, loosely based upon Dejerine's original clinical descriptions:

(1) hemialexia,
(2) alexia without agraphia,
(3) alexia with agraphia,
(4) aphasic alexia,
(5) pseudoalexia.

III. CLINICAL FORMS

1. *Hemialexia*

The presence of a unilateral reading disturbance (alexia) following section of the posterior part (splenium) of the corpus callosum has been reported by a number of investigators. The earliest such case was reported by Trescher and Ford (1937) in a patient whose posterior corpus callosum was sectioned by Walter Dandy for removal of a colloid cyst of the 3rd ventricle. These authors demonstrated that the patient was unable to recognize and comprehend words or letters presented to the left visual field alone despite the presence of fully normal visual fields. The authors conjectured that material presented to the left visual field reached the right visual cortex but that section of the splenium had effectively separated this area from the portion of the dominant hemisphere necessary for comprehension of written symbols.

Maspes (1948) reported on six cases with posterior callosal sectioning;

because of preoperative or operative cerebral damage to four of the cases he restricted observations to two individuals with no recognizable cerebral pathology except that produced by the callosal section. Both of these patients read and comprehended material presented to the right visual field but were unable to recognize or comprehend either words or letters presented tachistoscopically in the left visual field. More recently Gazzaniga *et al.* (1965), reporting several patients who had undergone complete callosal section, confirmed the earlier findings that the non-dominant visual field is alexic while reading is normal in the other field. Thus there appears to be a condition of hemialexia produced by surgical sectioning of the splenium of the corpus callosum. That the same syndrome may be produced on a vascular basis is suggested by the statement of Trescher and Ford (1937) that "one of us has observed several cases of alexia on the left in elderly patients having a vascular lesion in the distribution of the posterior cerebral artery but has not had the opportunity to determine the exact site of the lesion by post-mortem examination."

There is, however, a series of reports in the literature of individuals who did not develop hemialexia after callosal sectioning. Akelaitis (1941, 1944) reported 24 cases of callosal sectioning, nine of which included the splenium. In six of these reading was tested pre-and post-operatively and Akelaitis reported no significant change in reading ability, thus suggesting a bilateral cortical capacity for reading. Criticism of the Akelaitis findings (Maspes, 1948; Geschwind, 1965) has emphasized the special character of the patient population. With only a few exceptions this consisted of patients with severe cerebral pathology, present from birth or early infancy (four of the six reported cases were left-handed secondary to early severe left-hemisphere pathology). Geschwind also suggested that the presence of life-long seizure disorder might favor the use of cerebral pathways not normally employed. By contrast the cases of Trescher and Ford (1937) and of Maspes (1948) had 3rd ventricle tumors and had not developed seizures prior to surgery. A further criticism may be advanced that the testing of reading ability by Akelaitis (1941, 1944) was inadequate. Reading was tested only by the recognition of a large block letter (4.5 cm in height) placed in one visual field and left there until a response was given. The author states that for best results the letter was placed between 2 and 10 degrees lateral to the fixation point and he fails to report any

means of detecting eye movement. Thus his results are not comparable to those authors who used tachistoscopic presentation and leave open the possibility that the Akelaitis patients would also have had hemialexia if tested in this manner.

In a recent report, however, Gazzaniga and Sperry (1967) give some support to the Akelaitis view. They tested two patients who had undergone callosal sectioning by presenting single object names tachistoscopically to the left visual field only. The patients were unable to verbalize the word but pointed to the appropriate object with the left hand demonstrating comprehension of the *written* word by the right hemisphere. A third patient, however, failed consistently in this test. The differences between the patient who failed and those who succeeded may account for the apparent differences in performance of this task. The patient who failed was entirely normal until he sustained brain injury as an adult whereas, of the two successful subjects, one was twelve years of age and the other had probably sustained brain injury in early childhood. These findings suggest that the non-dominant hemisphere is capable of limited 'reading' in patients with early brain damage but not if damage occurs after dominance has been well established. This agrees with experiences in cases where hemispherectomy has been performed for infantile hemiplegia. Thus, while agreeing with the observations of Akelaitis, the work of Gazzaniga and Sperry also confirms the occurrence of hemialexia in the adult following sectioning of the corpus callosum if these is no history of early brain damage or childhood seizures.

There is further evidence that reading comprehension is not present to any significant extent in the nondominant hemisphere of most adults. As will be noted later most patients suffering from alexia without agraphia have a right hemianopia plus a lesion of the corpus callosum primarily involving the splenium. Testing the patients is simple since the right visual-field defect removes problems of fixation and the necessity for use of tachistoscopic presentation. These patients are not able to point to objects presented to them with either the right or the left hand when written names are presented to them.

In summary there is a condition in which a failure of reading comprehension (alexia) in one visual field (nondominant) follows destruction of the splenium of the corpus callosum. This is an understandably rare condition and can be termed hemialexia.

2. *Alexia without Agraphia*

The entity of alexia without agraphia has been recognized for many years and many reports appear in the literature, most of which consist of a single case, too often without autopsy correlation. There is great variation in the neighborhood signs which accompany the major symptoms. The constant features of all cases are the loss of the ability to read printed material with retained ability to write both to dictation and spontaneously. While the onset of this disorder is usually sudden it most often appears without other dramatic disabilities. In eight cases reported by Alajouanine *et al.* (1960) alexia occurred suddenly and without any significant forewarning. Occasionally the onset was accompanied by giddiness, vertigo, visual scotomata or other symptoms referable to the posterior circulation, but more often there was no significant symptomatology at the onset. Because of this many patients do not seek medical advice immediately and often are not seen by physicians for many months. Hinshelwood (1900) noted that as the loss of reading was the only symptom recognized by the patient, the ophthalmologist was often the first physician consulted.

The etiology of most reported cases of this type of alexia has been vascular, specifically thrombosis involving the posterior cerebral artery. There are no well-documented cases of alexia without agraphia produced by trauma or gunshot wounds, probably because the exact combination of dominant occipital and callosal lesions necessary to produce this syndrome is highly unlikely in a patient who survives major penetrating wounds. Alexia without agraphia has been produced by dominant occipital lobectomy (David *et al.*, 1955) but in most instances has been transient, probably because the surgeon does not section the splenium in performing this procedure. The occipital lobectomy must destroy many of the fibers coming through the splenium but probably not all. Obviously if all the fiber connections traversing the splenium were destroyed a permanent syndrome of alexia without agraphia could be anticipated.

A. *Clinical Symptoms*

Some symptoms are frequently found accompanying alexia without agraphia and many other symptoms are recorded in a few cases. A right homonymous hemianopia is most common and many authors state that this is an invariable accompaniment (De Massary, 1932; De Ajuriaguerra

and Hécaen, 1949), but there are several cases recorded in which no field defect was detected (Adler, 1944; Alajouanine *et al.*, 1960). A defect in the ability to identify colors on confrontation has been recorded in most cases. Defective ability to read musical notes or to read numbers has been recorded in many, but not all. In addition, a calculation disorder is frequently seen, and difficulty in naming objects and defect in topographical orientation are reported in some cases. Because the associated symptoms have so often led to theories concerning the mechanism of the alexia, the theories will be discussed together with the individual clinical signs. It is significant to note that paralysis, sensory loss, paresthesiae and significant aphasia are rarely recorded and are almost never noteworthy in cases of alexia without agraphia.

Alexia: The most striking symptom recorded is of course the inability to read printed material. Many authors (Dejerine, 1892; Hinshelwood, 1900; Hécaen, 1967) have divided this into two varieties, literal and verbal alexia. In addition some patients are described who were able to read letters, but unable to join them together to make syllables (asyllabia). Many authors have noted that most 'pure' alexics have difficulty with both letter and word reading and that none are completely intact in one performance while failing the other and consider literal and verbal alexia as points on a continuum. Alajouanine (1951) feels that almost all alexic patients have some limitation of the ability to recognize letters, many have difficulty in orienting the letters in space or in recognizing the letters as a graphic symbol, disturbances which interfere with the visual-auditory associations needed for recognition. Frequently used letters are recognized better than infrequently used letters, often the patient's own initials being best preserved. Simply designed letters such as 'I' and 'O' are recognized more readily than more complex letters. Errors also occur for letters such as 'P' and 'R', b', 'd', 'M' and 'W', etc. which are frequently substituted for each other. Dejerine (1892) reported an individual who could not 'read' the letters 'R' and 'F' until they were inscribed in a medallion signifying the French Republic at which time they were immediately recognized.

Some patients will read individual letters, place them together in syllables which are pronounced audibly and from this will decipher the word. If given adequate time some of these patients can comprehend

small quantities of written material. Usually, however, this process leads to both paralexia and omission so that comprehension of sentences and paragraphs is almost impossible. The alexic patient is often able to read some simple, high-frequency words more readily than he can identify the component letters. Hinshelwood (1900) considered this as a separate and specific disability which he termed 'letter blindness without word blindness'. This condition is rarely reported now, probably because most investigators note that in fact only a few words are actually identified by these patients who thus can be considered 'word blind' as well as 'letter blind'. There may, however, be a notable discrepancy in the ease with which an alexic handles letters and words; one patient may 'name' letters while unable to 'read' words while another patient may be unable to distinguish the separate letters of a word he is able to read aloud. This discrepancy has been emphasized in the recent classification of Hécaen (1967) who calls the first variety verbal alexia, the second literal alexia and uses the term global alexia to describe the patient unable to 'read' either letters or words.

It should be noted, however, that what might be considered as the syndrome of 'letter blindness without word blindness' is seen frequently in aphasic patients. Thus patients with classical anomic aphasia who manifest severe difficulty in confrontation naming may be able to read words (i.e. show comprehension of written words) but may be unable to name out loud individual letters just as they have difficulty producing the names of objects, body parts, etc. An important test of this would be matching of capital and lower case written and printed letters which should reveal normal performance and of course the presence of clinical aphasia excludes these individuals from the category of alexia without agraphia. We believe that these are cases of letter-naming difficulty rather than cases of literal alexia without verbal alexia and that some of the cases described in the literature as isolated literal alexia would fall within this description.

Writing: Most authors agree that the writing seen in alexia without agraphia is not entirely normal, but are impressed by the retention of considerable writing capacity compared to minimal reading ability. A frequent observation is that patients will write better to dictation or when asked to write a spontaneously formulated paragraph than in copying. This does

not mean that they cannot copy, but this performance is slavish and often without understanding. The behavior of such a patient is like that of normals attempting to copy a meaningless design. The fact that the patient can copy exactly although slowly words whose meaning remains unknown to him is important in ruling out a primary perceptual disturbance and favors a disturbance in an associative mechanism. The ability to transfer written script to printed letters or to block letters is extremely difficult or impossible for the alexic patient. Even spontaneously performed writing shows certain disturbances. Many authors have noted the inability of the alexic patient to continue writing after a brief distraction. Many of these patients have difficulty in moving from one line to the next and often the writing is slanted abnormally with the writing directed downward and to the right. Dejerine (1891) commented that the correct writing associated with poor following of printed lines resembles the written performances of a patient with acquired blindness. Purdon Martin (1954) found similar writing characteristics in an alexic patient and in normal individuals writing with their eyes closed. There are some reports of defective spelling but most authors believe that the written spelling of alexics is actually within normal limits. Adler (1944, 1950) extensively studied a single patient shortly after the onset of alexia and again five years later. There was a distinct deterioration in writing which she considered due to an inability of the patient to benefit from practice. There are, however, many other reports in which there is no deterioration of writing.

Musical note and number reading: There is far less agreement concerning the loss of the ability to read musical notation or the ability to read numbers. Some patients have been reported who have retained both of these abilities while totally losing the ability to read while other patients, equally well studied, have lost one or both of these faculties. Thus the patient of Dejerine (1892) retained the ability to read numbers but lost the ability to read music. It should be noted, however, that he was still able to perform musically, both vocally and instrumentally even though he no longer read music, a situation analogous to alexia without agraphia. De Ajuriaguerra and Hécaen (1949) state that musical blindness always accompanies verbal blindness but Bastian (1898) and others report cases of retained ability to 'read'

musical notation despite inability to 'read' letters. This problem is complicated by the comparatively high rate of musical illiteracy in the general population and by variable, usually inadequate, testing of musical notation comprehension. It would appear that musical note reading may be lost in cases of 'pure' alexia but whether this is universally true remains a debatable point.

The ability to 'read' numbers has been studied and recorded adequately in many cases. Many patients with alexia without agraphia have difficulty in 'reading' numbers (Hécaen *et al.*, 1957) but most investigators (Alajouanine *et al.*, 1960) feel that number reading is likely to be preserved, as in Dejerine's original case (1892), suggesting as explanations that: (1) the number vocabulary is smaller and more thoroughly overlearned, and (2) the phonemic qualities of numbers are less complex than those for letters. A third explanation points out that numbers are first learned by counting on the fingers, thus producing bilateral somesthetic associations to the written numerals and that therefore alternate association pathways may be available for number reading (Geschwind, 1962).

Calculating ability is often disturbed in patients with alexia without agraphia, again the severity ranging from no disturbance to total incapacity. Most authors are agreed, however, that acalculia per se is not a part of the alexic syndrome and if present suggests either a severe number-reading disturbance (De Massary, 1932), a spatial orientation disability or a combination of these. Dejerine's classic case (1892) provides a dramatic example in that his ability to read numbers and to calculate was normal despite a severe and persistent alexia.

Reading by nonvisual stimulation: (1) Somesthetic 'reading'. As early as 1877 Charcot reported a patient who could read only by using his finger to trace the outline of each letter, a finding that was strongly emphasized in the work of Goldstein and Gelb (1928). That this capability is not universal in alexia without agraphia is attested by other observers (Hécaen *et al.*, 1957; Alajouanine *et al.*, 1960). Nonetheless, the ability of many patients to 'name' a letter traced somesthetically after failure on purely visual stimulation is well documented. Similarly most patients with pure alexia can 'read' anagram letters held in the hand. Patients with alexia with agraphia are unable to make use of these somesthetic stimuli. (2) Auditory 'reading'. The patient with alexia

without agraphia readily comprehends words spelled aloud to him by the examiner and also spells aloud correctly while patients with other varieties of alexia fail on these tasks (see later sections for further discussion of this evaluation).

Color identification: A fascinating and frequent accompaniment of alexia without agraphia is difficult or even totally inability to identfy a color upon confrontation or to point to the proper color when the color name is offered. This defect is additionally striking because most of these patients can name objects without difficulty.

A color identification disorder was reported by Broadbent (1872), and has subsequently been reported frequently. In fact, defective color identification ranks in frequency just after alexia and right visual-field defect as a persistent finding in this syndrome. Not all cases of 'pure alexia', however, show defective color identification (Quensel, 1927; Davidenkov, 1956; Ajax, 1964).

The source of this color-identification defect remains controversial. One frequent suggestion is that this is a form of agnosia (Holmes, 1950; Hécaen *et al.*, 1957; Kinsbourne and Warrington, 1964), a theory that has been discounted by others (Bay, 1953; Critchley, 1953; Teuber, 1955). In a recent review Critchley (1965) stated that there are only a few cases of *color agnosia* recorded in the literature and that he did not feel that any of these were well studied. After further subdividion of the color identification problem he stated "...it seems likely that the syndrome of *amnestic color blindness* really represents not a specific entity, but rather a very mild disorder of visual perception coupled with a minimal aphasia." This theory was criticized recently by Geschwind and Fusillo (1966). These authors presented a case of alexia without agraphia who showed complete inability to name colors on presentation or to properly select colors when the name was given. This individual could, however, accurately match colors despite marked differences in brightness and saturation and had no difficulty in the Ishihara test. Similarly he had no difficulty handling color names on a strictly verbal level (i.e. he could state the color of familiar objects such as a rose, the sky, a banana, etc). He showed no evidence of anomic disturbance in other categories. These authors suggested that their patient's color-name disability was a loss of purely visual-verbal association, similar to the reading disability and

suggesting a specific disconnection as the cause (see later section on disconnection theory). Geschwind (1967) has pointed out the difference between this syndrome and the usual aphasic anomia. Because the patient fails both in naming a color and selecting a named color he suggests that it is best described as a failure to match a seen color and its spoken name. Recent work of De Renzi and Spinnler (1967) suggests that individuals with right brain damage have greater difficulty with nonverbal color identification tasks (color matching, Ishihara test) while those with left brain damage, as anticipated, have more difficulty in color-naming tasks. They point out, however, that their patients do not have the unique syndrome described by Geschwind and Fusillo and the possibility exists that the color-identification difficulty seen frequently in alexia without agraphia differs fundamentally from other color-naming and color-matching defects of brain-damaged patients.

Visual agnosia: Just as agnosia is often advanced as an explanation for the difficulties of color identification seen in alexia, it is also offered as an explanation of the alexia itself (Adler, 1944; Holmes, 1950; Gloning *et al.*, 1955; Ettlinger and Hurwitz, 1962). The topic of visual agnosia is complex and beyond the scope of this chapter (see Leischner, 1969), but does deserve comment because it has been implicated as the cause of 'pure' alexia.

Unfortunately the term agnosia has been used in many different ways in the literature. Some use the term in a broad clinical sense to describe any failure in recognition tasks with preservation of elementary sensation. In this broad sense alexia can be accepted as a form of agnosia. Many investigators, however, also use the term agnosia as an explanation or mechanism for a specific behavioral abnormality, defective symbol recognition. In some cases this is only circular thinking, i.e. alexia is a form of agnosia and is therefore caused by agnosia. Others use the term agnosia to designate a disturbance of higher-level perceptual functions, i.e., they use the term agnosia for the disability that Lissauer (1889) designated 'apperceptive mind blindness' and suggest that alexia is based on this difficulty. This is open to question, however; most alexics can copy adequately (an act that would seem to demand perceptual integrity). Furthermore, there are reports of patients with severe object-identification difficulties who can read (Davidenkov 1956). Quite possibly there are alexics with an apperceptive form of agnosia (Gloning *et al.*,

1955) but this does not appear to be a factor common to most cases. Finally, some investigators consider alexia to be agnosia of the associative type (in the terminology of Lissauer), i.e., a loss of the usual associations of the seen name. Thus agnosia as presently used appears too vague for the explanation of alexia unless the process producing this state is well described.

Some authors describe alexia as an agnosic phenomenon in a different sense, arguing that the difficulty in reading is part of a more wide-spread difficulty in visual identification. There are some recorded cases of alexia which have been accompanied by additional findings of defective visual recognition. A good example is the case of Adler (1944) in which the patient had alexia with minimal writing disturbance plus inability to identify objects, pictures of objects, faces and geometric shapes. In this situation the inability to 'recognize' letters and/or words can be considered part of an overall defect in symbol recognition although the precise mechanism of this defect remains obscure. It is well recognized, however, that many cases do not have this combination of findings; some patients are alexic without agnosia and others agnosic without alexia (Hécaen *et al.*, 1957). In this respect two cases reported by Davidenkov (1956) are illustrative. The first patient, following a vascular accident, was able to read and write but could not recognize (name) common objects on visual presentation but readily named these objects when he was allowed to touch them. The second individual lost the ability to read although he retained full capacity for writing and had only minimal difficulty in naming objects (or colors) visually presented. Davidenkov emphasized that the first case was one of visual agnosia and was quite distinct from the second, a case of pure alexia with minimal evidence of 'visual agnosia'. The opinion offered by Davidenkov is shared by many other investigators who have noted that recognition (naming) of visually presented objects is retained in most cases of alexia without agraphia. Thus, while pure alexia and visual agnosia (in the sense of dificulty in identification of nonverbal visual material) certainly can coexist they may also occur separately.

In summary, we feel that the term agnosia is not useful as an explanation for the occurrence of alexia without agraphia and that its use should be restricted to those cases which show widespread difficulties in visual identification including the comprehension of written material.

Defective Gestalt formation: Some authors argue that alexic patients suffer from a defect in Gestalt formation, an inability to combine the parts into a whole. With this disorder the patient is able to recognize parts of the written material but unable to combine them into a meaningful whole or vice versa, reads words without being able to isolate (read) letters. This explanation was particularly stressed by Wolpert (1924, 1930), Goldstein and Gelb (1928), Brain (1941), and Adler (1944). Unfortunately the Gestalt theory neither defines nor explains the disturbances underlying alexia. Furthermore, many patients with right hemisphere lesions (see later section on unilateral paralexia) seem to have difficulty in uniting parts into a whole but reading is usually less markedly disturbed than other visual functions in these patients, suggesting that defective Gestalt formation is not a basic mechanism in the production of alexia.

Simultanagnosia: Several authors (Wolpert, 1924; Davidenkov, 1956; Luria, 1959; Kinsbourne and Warrington, 1962b) have reported patients manifesting reading disability ('spelling dyslexia') who are able to describe a single item from a complex picture but not other items in the picture and thus cannot recognize the story portrayed by the entire picture. This has been called simultanagnosia or a disorder of simultaneous form perception. The patients described were able to 'read' single letters and thus laboriously decipher simple words but were both slow and liable to erroneous guessing (paralexic substitutions). The obvious similarity between identification of a single portion of a complex picture and a single letter of a word was duly noted and the 'spelling dyslexia' was considered a natural consequence of simultanagnosia. Through tachistoscopic presentation of both single and double forms Kinsbourne and Warrington (1962b) demonstrated that their patients with this disorder were unable to identify more than a single stimulus. They also demonstrated a need for a minimum of one second between exposures of two different forms for these to be properly identified by such patients. These authors suggested that these two observations were sufficient to 'explain' simultanagnosia and spelling dyslexia and suggested dominant anterior occipital localization. This inviting theory cannot be considered in most cases of alexia without agraphia, however, as most reports describe no such defect in the comprehension of complex pictures and do not show

'spelling dyslexia'. This latter point is best demonstrated by the ability of many alexic patients to copy an entire word without identifying either the letters or the word. Thus simultanagnosia can be considered a source of alexia without agraphia, but only in selected cases.

Eye-movement disorder. Several authors have noted disturbances of eye movements in patients with reading difficulty. Luria (1966) includes a careful study of eye movement in his tests of reading ability. Beringer and Stein (1930) reported an individual who would read one or two words, stop, blink his eyes and then read several more words. In addition, this patient had extreme difficulty in changing from one line of print to the next. The authors suggested that the reading disturbance was due to a combination of visual defect (right hemianopia) producing a need for increased stimulation of remaining visual tissue and an increased refractory phase of the remaining retinal tissue. The authors believed that with visual stimulation there would be slow and uneven stimulation of the visual cortex, abnormal motor response (eye-movement disorder) and subsequent reading difficulty. Warrington and Zangwill (1957) present a single case, unable to read words, but capable of reading individual letters. This patient had a persistent right visual-field defect, distinct abnormalities of saccadic and searching eye movements plus a tendency to read correctly the left portion of a multisyllable word and to guess the remainder (e.g. 'them' for 'these', 'manager' for 'management'). They considered that eye-movement disorder could produce a disability in word reading, but did not explain difficulties in letter reading which they considered a true agnosia (defect in symbolic recognition). This theory fails to explain the absence of alexia in patients with severe paralysis of ocular movement. Although disturbances of eye movements do occur in some cases of alexia it would appear logical that alexia itself could produce abnormal eye movements just as visual scanning of an unfamiliar form is less regular than the visual movements used to scan a familiar object. Thus the possibility must be raised that the alexia produces eye-movement irregularities rather than vice versa.

Asthenopic alexia: Pötzl (1928) describes a specific type of alexia without agraphia seen in patients who have had vision restored surgically after prolonged blindness. He termed this asthenopic alexia and noted that

this was always a transient state. These patients can read for only a brief period and then must rest before reading further.

Geometric form agnosia. Spatial disorientation: A number of patients with alexia without agraphia have shown abnormality of spatial perception. That this is not constantly seen was pointed out by Charcot (1887) and Dejerine (1892), but the role of spatial perception disturbance as an etiologic factor in reading disturbances must be considered. Purdon Martin (1954) suggested that many alexic patients have defective visual-spatial perception. These patients have difficulty in drawing a line between dots or in defining the relationship of two lines. This author noted the similarity in these performances to those of a patient with a refractive error who would be unable to read without his glasses and whose performance is spatially oriented drawing tasks would be grossly hampered. Gloning *et al.* (1955) described a right-handed individual who suffered a right occipital lesion and subsequently had 'pure word blindness', visual-spatial agnosia, constructional apraxia and inability to read a clock. They considered that the reading disturbance in their case was caused by the severe visual-spatial agnosia and was not, therefore, the same as the alexia noted with left occipital defect. Adler (1944) also reported a marked geometric visual difficulty in her patient, but concluded that this, as well as the alexia, was on the basis of a figure-ground disturbance. Geschwind and Fusillo (1966) reported a case of alexia without agraphia who originally showed disorientation in space but recovered from this completely while the alexia persisted.

B. *Pathology*

Despite the large number of cases quoted in the previous paragraphs, alexia without agraphia remains an uncommon condition although reported more frequently than pure word deafness or isolated agraphia. Most papers in the literature report only a single case. One of the authors (F.B.) has observed only a single case of 'pure' alexia in somewhat over 1000 consecutively evaluated cases of language disturbance. Thus most neurologists, even those with large clinical practices and specialized interest in higher cortical dysfunction, cannot anticipate seeing more than a few cases of alexia without agraphia in a lifetime of practice.

The rare occurrence of this syndrome is amply substantiated by the

infrequent appearance of autopsied cases of alexia without agraphia. Table I lists 17 cases taken from the literature, each with a clinical diagnosis of alexia without agraphia and a description of the neuropathology. Unfortunately the completeness of both clinical and pathological reports varies widely; the omission of at least some pertinent information in one or both categories occurs in many of the cases. Nonetheless, the basic clinical picture appears solidly established in each case and sufficient pathological data are included to offer useful comparisons. The pathological findings will be discussed under three headings: occipital, corpus callosum and other involvement.

Occipital: With only one exception the left occipital lobe was involved in all cases. In the exceptional case (Gloning *et al.*, 1955) the right occipital lobe was involved but the authors strongly suggested that the reading disturbance in this case was produced by visual-spatial distortion and is not the same as that seen in 'pure word blindness' produced by left occipital pathology. The description of the area involved is somewhat variable but usually includes the inner and under surface of the occipital lobe, essentially the area directly dependent upon the posterior cerebral artery for vascular supply. Most often the lingual and part or all of the fusiform lobe are infarcted along with varying portions of the cuneus and the calcarine cortex. One report (Bonvicini and Pötzl 1907) stated that the calcarine cortex appeared intact although surrounded by infarcted cortex. Dide and Botcazo (1902) and Kleist (1934) describe bilateral occipital infarction but the remainder describe unilateral involvement.

Splenium of the corpus callosum: Infarction or 'involvement' of the corpus callosum or of the 'connection of the right visual area' is described in nine of the fifteen brains. This does not mean, however, that actual infarction was not present in the other cases. Infarction in the tightly packed white matter of the splenium may not produce the grossly obvious degenerative changes that are seen after involvement of cortical and subcortical tissues. One of the present cases (Geschwind and Fusillo, 1966) was sectioned by a highly experienced neuropathologist who reported no evidence of corpus callosum infarction on gross inspection. Sections stained for myelin demonstrated, however, a pronounced destruc-

TABLE I

Published cases of alexia without agraphia with autopsy findings

(1) Landolt (1888)	Old cystic lesion involving the inferior portion of the left occipital lobe. Recent hemorrhagic lesion in the corpus callosum.
(2) Dejerine (1892)	Old lesion in left occipital lobe and splenium of corpus callosum. Fresh lesion in left angular gyrus.
(3) Redlich (1895)	Lesion involving left calcarine, lingual and fusiform cortex, splenium and posterior thalamus.
(4) Lissauer (1889) }	Degeneration in left medial occipital lobe and
(5) Brissaud (1900) }	splenium.
Hahn (1895)	Softening in left calcarine region, splenium and right tapetum.
(6) Dide and Botcazo (1902)	Bilateral lesions of lingual gyrus.
(7) Hinshelwood and MacPhail (1904)	Old lesion, left calcarine fissure.
(8) Paterson and Bramwell (1905)	Lesion in the distribution of the left posterior cerebral artery including connections of the right visual areas with left angular gyrus.
(9) Bonvicini and Pötzl (1907)	Two lesions in the left occipital cortex which enclosed an apparently intact calcarine cortex.
(10) Pötzl (1928)	Destruction of left lingual, fusiform and part of cuneus, some degeneration in left parietal and temporal lobes, destruction of part of corpus callosum.
(11) Ehrenwald (1929–30)	Old lesion involving the left lingual gyrus.
(12) Kleist (1934)	Inner and under surface of left occipital lobe, small right occipital lesion with callosal degeneration.
(13) Niessl Von Mayendorff (1935)	Left lingual and fusiform gyri, cuneus, precuneus and tapetum.
(14) Gloning *et al.* (1955)	Right occipital (lingual, fusiform and lower cuneus) splenium and posterior thalamus. Recent left frontal and minimal left occipital necrosis.
(15) Geschwind and Fusillo (1966)	Left calcarine region, splenium of corpus callosum, VPL of left thalamus and left hippocampal region.
(16) Gloning *et al.* (1966), case 1	Meningioma, deep into left medial occipital lobe with considerable surrounding degeneration.
(17) Gloning *et al.* (1966), case 2	Cystic lesions in left lingual and fusiform gyri and splenium.

tion of the splenium. This does not mean, however, that an infarct of the splenium is essential for production of the syndrome of alexia. Several authors (Kleist, 1917; De Ajuriaguerra and Hécaen, 1949) have suggested that deep white-matter destruction in the left medial occipital region could just as readily disconnect the left-hemisphere language areas from right-visual sensory stimuli.

Additional involvement: In addition to the universal presence of occipital pathology and frequent splenium involvement in cases of alexia without agraphia several areas of inconstant involvement are mentioned. Most of these structures fall within the area of distribution of the posterior cerebral artery. Thus some cases showed posterior thalamic or hippocampal infarction (producing thalamic pain or disturbance of recent memory). Three had parietal softening although in two of these the parietal lesion occurred later than the occipital involvement. Most authors report that the remainder of the brain and brainstem were free of significant pathology. Thus the only consistently demonstrated pathology involves the dominant occipital lobe and the connections of the non-dominant visual association area with the dominant speech areas.

All of the reports listed in the table describe medial-inferior occipital involvement but a number of postsurgical cases of alexia without agraphia secondary to lateral parieto-occipital pathology have been recorded (Warrington and Zangwill, 1957; Kinsbourne and Warrington, 1964; Ajax, 1967). The pathology in these cases was diverse including tumor, A-V malformation and intracerebral hematoma. The exact depth of occipital involvement remains unknown as no postmortem follow up is available. As could be expected this type of case has a greater number of demonstrable neurologic side effects (mild aphasia, mild agraphia) but does show a distinct reading disturbance. Clinically these cases more closely resemble the cases of alexia without agraphia following dominant occipital lobectomy reported by David *et al.* (1955). The surgical cases, however, have either been transient or when persistent the reading disturbance has been mild. It is worth noting, however, that even following recovery of reading ability the surgical patients usually dislike the reading process. It would be possible to suggest that the fundamental pathology in these cases is also in the medial-inferior occipital region but this can not be confirmed by the available material. The possibility of a

second anatomical localization for the production of reading disturbance without agraphia can be raised but at best must remain conjectural until better clinical and pathological proof is presented.

C. *Theoretical Considerations*

Many of the symptoms associated with alexia without agraphia discussed above have also been suggested as possible causes of the alexia. These include visual agnosia, simultanagnosia, defective Gestalt formation, eye-movement disorder and visual-spatial disorientation; inasmuch as these have already been discussed they need not be reiterated but one theory, that of *disconnection*, has not been brought forward and deserves additional attention.

Several authors have suggested that alexia without agraphia is produced by separation of two essential cortical areas (Dejerine, 1892; Hinshelwood, 1900; Nielsen, 1946; and more recently Geschwind, 1962, 1965). These investigators have suggested that alexia seen without other major language disturbances represents an inability to transfer visually perceived language stimuli to an area in the left hemisphere involved in interpretation of visual language (angular gyrus). This theory was advanced by Dejerine (1892) who pointed out that the primary visual area in the left hemisphere is damaged (thus causing the right visual-field defect) and that in addition there was destruction of connections from the right visual region (either by a lesion in the corpus callosum or in the white matter deep to the left parietal-occipital region) which effectively disconnects the dominant angular gyrus from the visual association areas of both sides. This theory proposes that the area for written language interpretation is intact, thus the patient can recognize spelled words and can spell accurately both orally and in writing. The separation of this area from visual stimuli r only produces alexia but also can cause a color-identification disturbance which is also dependent on pure visual-verbal associations. Most of these patients, however, retain object-naming ability and many can correctly 'read' numbers, possibly through other pathways available for these visual associations.

3. Alexia with Agraphia

The clinical syndrome and pathology of alexia with agraphia were presented by Dejerine (1891) whose case manifested almost total inability

to read, limited writing ability, but only minimal aphasia and calculation disturbance. At postmortem examination Dejerine found a yellowed plaque confined to the angular gyrus on the left side with penetration into the white matter. Dejerine stressed that this syndrome must be carefully distinguished from 'pure word blindness' (i.e. alexia with no difficulty in writing). The clinical picture of alexia with agraphia has been described many times since the work of Dejerine and most investigators have agreed that the angular gyrus is essential for the functions of reading and writing (Wolpert, 1930; Nielsen and Raney, 1938; Alajouanine *et al.*, 1960).

This entity was also seen frequently before the time of Dejerine's lucid clinical description. For example Starr (1889) collected 50 cases of 'sensory aphasia' from the world's literature, 21 of which had proved angular gyrus lesions at autopsy. All of these had demonstrable alexia. Unfortunately many of the examiners had not recorded or tested writing, but whenever tested, writing was also noted to be abnormal and the occurrence of both reading and writing disturbance following damage to the dominant angular gyrus was well accepted by that early date.

Alexia with agraphia has been designated by many other terms. Some authors speak of this symptom complex as 'parietal alexia' (Quensel, 1927; Pötzl, 1927; Hoff *et al.*, 1954); in the classification of Kleist (1934) mentioned previously this entity was called 'cortical alexia'. Other authors (Nielsen, 1946; Alajouanine *et al.*, 1960), have considered this syndrome as one of a group called 'aphasic alexia'. A number of authors have pointed out that alexia with agraphia is a total defect in the use of written or printed symbols and have claimed that this suggests a 'language defect' rather than a perceptual defect.

A. *Clinical Symptoms*

There is greater variation in the clinical picture of this syndrome than in alexia without agraphia. Many cases begin acutely with sudden loss of the ability to read and write as a result of an acute infarction. This disorder has also been reported with neoplastic disease and following trauma; there are several fairly large series secondary to war wounds (Marie and Foix, 1917; Poppelreuter, 1917). Alexia with agraphia has been described on the basis of an A-V malformation (Casey and Ettlinger, 1960) and tumors with left parieto-occipital localizations. While a variety of etiolo-

gies may produce this syndrome, most authors agree that involvement of the dominant angular gyrus is a constant finding. The wide variation in pathological causation contrasts significantly with alexia without agraphia (the permanent cases of which are nearly all of vascular origin) and helps make alexia with agraphia a far more common entity.

The clinical onset of alexia with agraphia may be insidious or quite acute. Almost invariably there are neighborhood clinical signs of significance. If the onset is acute the patient will usually show a mild paresis and some degree of sensory loss. The paresis clears, either completely, or at least to the point where only minimal weakness is demonstrable, but in many cases a significant sensory disturbance involving the right side of the body remains. Almost all patients suffering alexia without agraphia have some degree of aphasia which ranges from a minimal degree of word-finding difficulty to a more marked sensory aphasia with paraphasia and comprehension disturbance. It should be emphasized, however, that the term alexia with agraphia can be used only when the reading and writing disturbances are distinctly more severe than any other aphasic difficulties. In addition, the findings of the Gerstmann syndrome are often present. A right homonymous visual-field defect may be present but there are many reports (De Massary, 1932) where this sign is absent and it is clearly a less constant finding than in alexia without agraphia.

Alexia: The loss of the ability to read is almost invariably severe in cases of alexia with agraphia. The reading disturbance usually includes words, letters, numbers and musical notes although there are some exceptions to the latter two categories (e.g. Casey and Ettlinger, 1960). Not only is number reading frequently disturbed, but very often there is a severe calculation defect so that the patient cannot handle numerical computation even when the problem is given verbally and a verbal response is requested, a notable contrast with the usual situation in alexia without agraphia. Dejerine (1892) and De Ajuriaguerra and Hécaen (1949) point out that patients with this type of alexia are unable to trace letters or numbers with their finger and understand the symbol. While this capacity is not consistently preserved in 'pure alexia' these authors emphasize that the recognition of letters or numbers as meaningful symbols regardless of modality of presentation is invariably disturbed in cases of alexia with agraphia. The difficulty in somesthetic 'reading' is parallelled by difficulty

in auditory 'reading' (i.e., the inability to understand spelled words).

Agraphia: As the name suggests, writing disturbance is a distinct part of alexia with agraphia, but there is considerable disagreement as to the severity of the writing disturbance. Dejerine and many subsequent French investigators have stated that writing is less disturbed than reading. Other authors, however, find that reading and writing disturbances are approximately equivalent so that the individual who is able to read a few letters or a few words will also write a few letters or a few words. The writing disturbance is usually not so severe that the patient is unable to produce letters, but the writing is always paragraphic, consisting of recognizable letters combined into unrecognizable words. While these patients spontaneously produce correct letters they are most often unable to produce letters upon dictation or will be paragraphic in this situation. These patients often show significant disturbances in copying printed material in contrast to patients with alexia without agraphia who copy slowly and slavishly but correctly while the alexic with agraphia will often reproduce the actual forms of the letters incorrectly.

Gerstmann syndrome: In addition to a calculation defect which appears on verbal testing and thus goes beyond the inability to read and write numbers, these patients often show the other findings making up the Gerstmann syndrome (Gerstmann, 1930). Thus many patients with alexia with agraphia have difficulty in right-left orientation and difficulty in naming fingers or recognizing them by name along with agraphia and acalculia. These symptoms are not invariably present and are, in fact, rarely mentioned in the literature. Our own experience has been that most patients showing the syndrome of alexia with agraphia will have an associated Gerstmann syndrome.

Related symptomatology: Many of these patients show other symptoms which are seen frequently in conjunction with the Gerstmann syndrome. Thus most of these patients have some form of constructional disturbance, showing distinct difficulty in copying drawings, performing match stick designs or Koh's Block Designs. Topographic memory disturbance (Critchley, 1953) is not infrequent even when evaluated in a purely visual (nonverbal) manner. Many cases have been reported showing inability

to recognize or locate an area on an outline map. It has been our experience, however, that most of these patients are oriented in their own and even new environments and severe spatial disorientation is not a frequent accompaniment of this type of alexia.

Aphasia: Some form of aphasia is almost constant with this syndrome and, as noted above, is most pronounced if the onset is acute. The aphasia is fluent and in most cases the aphasic disturbance is mild, consisting primarily of somewhat empty speech and word finding pauses, difficulties in naming, usually in association with good repetition and auditory comprehension. In other words, these patients often manifest a mild anomic type of aphasia. In the classification of alexia proposed in this chapter individuals with severe aphasic disorders are considered as suffering from aphasic alexia rather than alexia with agraphia. Thus for a patient to be included under the term alexia with agraphia he must show much greater disturbance in reading and writing capacities than in spoken language functions. Some authors (De Ajuriaguerra and Hécaen, 1949) believe that alexia with agraphia is always accompanied by a distinct disturbance of inner speech mechanisms. We have seen, however, many patients with severe reading and writing disturbance who are not functionally disabled in spoken language capability despite the presence of mild aphasic symptoms.

Spelling: Geschwind (1962) and Howes (1962) have laid great stress on the inability of these patients to comprehend words spelled aloud by the examiner despite the presence of otherwise excellent auditory comprehension. The reverse task, the spelling of words out loud is also severely defective. This is in sharp contrast to the patients with alexia without agraphia who generally have little difficulty in either of these functions. Thus we believe that an excellent ancillary test for this particular variety of alexia is to ask the patient to spell aloud or to identify a series of words spelled aloud by the examiner. Of course cases with more significant aphasic defects, particularly of the sensory aphasia variety, will also fail this test but in this situation the spoken language disturbance is sufficiently prominent to explain the failure in the spelling tasks.

Some investigators regard this difficulty in comprehending words spelled aloud as evidence of a mild degree of auditory comprehension

difficulty, but this view is difficult to maintain in the face of evidence that patients with this disorder may show absolutely normal comprehension for long and complex sentences. Other authors (Symonds, 1953) regard this as a specialized auditory comprehension defect which they call 'letter-name deafness'. We believe that a simpler explanation is available. As noted earlier, the alexic without agraphia is able to 'read' when letters are presented tactilely or auditorily while the alexic with agraphia fails in both these situations. The alexic with agraphia is in fact much like a patient returned to a state of illiteracy. One might almost say, by analogy to discussions of 'inner speech' that 'inner reading' is preserved in alexia without agraphia but lost in alexia with agraphia. The ability to comprehend orally spelled words requires ability to turn the heard letters into their visual form, an ability never acquired by the illiterate and lost in alexia with agraphia.

Paralexia: In some patients with long-standing alexia with agraphia an unusual form of paralexia may be noted. These patients are unable to comprehend most written material. Occasionally, however, when attempting to read aloud, they will produce responses which show comprehension of the word. These responses are often paraphasic substitutions. Thus one of our patients read the word 'kitten' as 'a small cat'. When asked to read the word 'livingroom' she said, "Oh, I know what that is, it is the place we go after dinner to watch TV." It should be stressed that this patient had a significant defect of naming (word finding) on visual confrontation. It appears possible that certain written words aroused visual images which were then named paraphasically. Obviously this type of 'reading' is possible only for picturable words and she was unable to respond to written prepositions, most verbs and abstract nouns (e.g. liberty, truth, etc.). Also, the stock of correct responses was limited and had taken many years to develop so that this individual, while comprehending a few written words, would still be considered alexic.

B. *Pathology*

Dejerine (1891) reported postmortem findings of an old, yellowed infarct involving about three quarters of the left angular gyrus as the only significant pathological finding. From this base the author surmised that

the angular gyrus was essential for the optic recall of written letters and that damage to this structure produced both alexia and agraphia. This pathological localization has been almost universally confirmed and it is generally accepted that destruction of the dominant angular gyrus will cause significant disturbance in both reading and writing (Wolpert, 1930; De Massary, 1932).

C. *Theoretical Considerstions*

Several interpretations have been placed on this syndrome. It is difficult to accept the notion that this is simply a more severe degree of alexia without agraphia since the differences in clinical picture and pathology militate against the notion that these are disturbances distributed on a continuum. It is also not possible to accept the idea that this is simply a portion of a more widespread aphasic syndrome since the reading and writing disturbances may clearly dominate the clinical picture. Dejerine (1891) felt that this syndrome resulted from an actual lesion of the cortical center for written words. The effect of the lesion may be roughly considered as reducing the patient to a state of illiteracy. In what way does the angular gyrus subserve this function? Although it is clear that it is a vital link in the process of reading this does not clarify the fundamental activity of this region. The suggestion has been made recently (Geschwind, 1965) that the angular gyrus plays a major role in the formation of associations between vision, somesthesis and audition and thus is basic to the visual-auditory associations involved in reading. While the theoretical problems remain unsettled clinical experience clearly demonstrates the important status of the dominant angular gyrus in written language function in that significant angular gyrus damage can produce both alexia and agraphia with comparative sparing of spoken language function.

4. *Aphasic Alexia*

Most investigators of aphasic syndromes have described reading disturbance associated with aphasia. Many agree with De Massary (1932) who stated that significant aphasic symptoms may produce so much disturbance of language that reading is secondarily involved. He concluded that such cases belong properly in a classification of aphasia and not in an outline of alexia. Nonetheless, certain characteristic reading disturbances are present in some forms of aphasia and will be outlined

briefly. It should be noted that one of the greatest difficulties in outlining the aphasic alexias stems from the diverse classifications of aphasia. Most of these classifications actually describe the same or significantly similar symptom complexes but use different terminologies. We have elected to use the terminology presently in use in our unit modified from the classic outlines of the early continental investigators.

A. *Wernicke's Aphasia*

In this type of aphasia the patient does not understand spoken language or shows severe defect in comprehension of spoken words. His speech is usually fluent, but filled with paraphasia. Repetition of spoken language is severely disturbed and most patients with this type of aphasia have extreme difficulty in producing names of objects on confrontation. These patients have an alexia accompanied by agraphia of a variety similar to that described under the category of alexia with agraphia, in other words a sprawling paragraphic output made up of real letters combined in an incorrect manner. The defects in reading and writing are generally of the same order of magnitude as those seen in producing and comprehending spoken language.

It is generally accepted that the pathology producing Wernicke's aphasia is located in the posterior superior temporal lobe of the dominant hemisphere. This area lies directly adjacent to the angular gyrus and shares at least some of the same vascular supply. Thus it would be easy to conjecture that the alexia and agraphia seen with Wernicke's aphasia is secondary to involvement of the angular gyrus by the same pathological process. It is true that such pathological involvement is present in many cases. There is, however, reasonable evidence that angular gyrus involvement is not essential and that posterior superior temporal pathology alone can produce severe alexia. Nielsen (1939) presented 16 cases, primarily collected from the literature, with clinically significant alexia who at postmortem had dominant temporal lobe infarction without involvement of the angular gyrus. Each of these individuals, of course, demonstrated the other features of Wernicke's aphasia.

There is a further argument against the theory that the alexia and agraphia of Wernicke's aphasia are secondary to extension of the lesion to the angular gyrus. This theory would carry the implication that the full syndrome of Wernicke's aphasia actually consisted of separate

components. It is, however, a commonplace of clinical experience that while alexia with agraphia may be seen in relative isolation, rarely or never does one see severely fluent and paraphasic speech together with auditory comprehension disturbance in the absence of alexia and agraphia.

The lesion producing Wernicke's aphasia lies in a region of auditory association cortex. Wernicke (1874) felt that the syndrome was not a mixture of separable components but that all the elements resulted from a lesion of the area subsequently given his name. It should be noted that the area described by Wernicke was confined to the posterior superior temporal region. Some recent authors (e.g. Penfield and Roberts, 1959) have tended to enlarge Wernicke's area to include all the retrorolandic speech regions. This nomenclature is, however, not justified historically, and appears not to coincide with either anatomical or clinical data. Wernicke argued that language is initially acquired auditorily in the great majority of mankind and that subsequent acquisition of visual language is by reference to this initial auditory acquisition. This would correspond with the clinical data that visual language may be disturbed in isolation by a lesion which prevents visual-auditory associations to be made while lesions in the cortical areas involved in this auditory learning will affect all modalities of language.

B. *Pure Word Deafness*

By definition the patient with this disorder is able to read with little or no difficulty whereas he does not understand spoken language. Similarly the patient with pure word deafness has at most mild difficulty in speech or writing although at times he may have slightly paraphasic speech and some paragraphic errors may be present in his writing. The classically accepted lesion for this entity lies deep in the dominant posterior temporal lobe separating the auditory association cortex from Heschl's gyrus but not involving the cortex itself or bilateral lesions in the middle of the superior temporal gyrus (Gloning *et al.*, 1963).

C. *Broca's Aphasia*

Loss of the ability to read is not anticipated in this type of aphasia, one in which the patient has no apparent difficulties with comprehension of spoken language. Many investigators, however, have noted reading disturbance in cases of Broca's aphasia (Nielsen, 1938; Goldstein, 1948;

Leischner, 1957). Nielsen labeled this as "one of the unsolved problems of aphasia" when describing ten cases taken from the literature, each with Broca's aphasia secondary to a purely frontal lobe lesion as demonstrated by autopsy and each said to be alexic. Review of the cases quoted by Nielsen is disappointing, however, as the evidence documenting loss of reading ability was poor. Several of the cases lacked evidence of premorbid ability to read (an important factor in the 19th century when illiteracy was common) and in others the ability to read aloud was not distinguished from reading comprehension. Certainly none of the cases quoted had the extensive clinical evaluation given to the patients with alexia mentioned thus far.

We would surmise that most cases reported as alexic with Broca's aphasia are explained in one of the following ways: (1) the patient was illiterate, (2) the patient was unable to read aloud, or (3) the patient suffered multiple lesions, one of which was posterior. We cannot exclude that true alexia may exist with a purely anterior lesion but evidence for this occurrence is not convincing. The possibility exists that a transient alexia may be present after frontal damage as a secondary effect of transient visual inattention such as described by Kennard (1939) and Welch and Stuteville (1958) in animals. Another possibility was raised by Charcot (1887) who thought that the poorly literate would be more likely to read aloud and could thus be expected to become alexic with onset of a Broca's aphasia. This theory has not, however, been subjected to careful investigation and remains unconfirmed.

We have had a limited experience with reading difficulties in patients with Broca's aphasia. Some of these patients had evidence for additional posterior lesions. In others the disturbance of reading was not entirely similar to that seen in the other forms of alexia. It was found that these patients could, in fact, comprehend written material but did so with tremendous difficulty, often needing several minutes to 'read' a ten or twelve word sentence. These patients disliked reading, considering it very hard work, but most of them, if directly questioned, stated that they were able to read, a view confirmed by formal testing. It would thus appear that the clinical evaluation of such patients demonstrates preserved reading comprehension. At present there is no adequate explanation for the slowness and dislike of reading in some patients with Broca's aphasia.

D. *Transcortical Aphasia (Isolation of the Speech Area)*
This rare entity is characterized by the presence of normal or near normal ability to repeat spoken language coupled with severe auditory comprechension defect and grossly abnormal expressive speech. Of the few patients described in the literature with this disorder (Goldstein, 1917; Geschwind *et al.*, 1968) all have been totally alexic and agraphic. It was theorized by Goldstein that in the isolation syndrome the auditory area of the posterior superior temporal region, the motor speech area in the posterior inferior frontal region and fibers connecting these two areas are intact but are, in essence, isolated from the remainder of the cortex, a view confirmed by the pathological findings of Geschwind *et al.* Thus while the patient is able to repeat words and phrases that he hears he does not comprehend them as words and is totally unable to recognize or comprehend printed material.

E. *Conduction Aphasia*
This type of aphasia, originally, although incompletely defined by Wernicke (1874), is not rare. The patient is able to comprehend spoken language, but unable to repeat correctly what he hears. In our own experience we note that these patients have a haltingly fluent speech, many literal paraphasias, good comprehension of spoken language, poor ability to name on confrontation and marked disturbance of repetition. In addition they cannot read aloud, but the great majority comprehend written material readily. Thus we have seen patients who cannot read a simple four- or five-word newspaper headline out loud but are able to read novels or even textbooks of medicine with apparently normal comprehension. They have difficulties in writing which parallel those of speaking. The written material produced by patients with conduction aphasia consists of real letters and even real words but is contaminated with misspellings (literal paragraphias) and omissions. In a small minority of patients who otherwise manifest the syndrome of conduction aphasia there is significant difficulty in reading comprehension despite good auditory comprehension. We have hypothesized that these patients have a posterior extension of their lesion from the parietal operculum (the usual site of the lesion of conduction aphasia) to the angular gyrus.

4. *Pseudoalexia*
In the discussion of the ability to read printed material there are several

varieties of difficulty which are not truly alexias but may present clinically as a severe difficulty in reading. Since they must be considered in the differential diagnosis of alexia they deserve comment here.

Reading aloud: The differentiation between the ability to read out loud and to comprehend written material is essential in any discussion of aphasic alexia. This has been emphasized by Goldstein (1948) and recently by Luria (1966) who note that many patients with aphasia in spoken language will have great difficulty in reading out loud and yet may have normal or near normal comprehension of written language. This will be true in particular of cases of conduction aphasia and Broca's aphasia. The opposite situation, i.e. the preservation of reading aloud in the face of inability to comprehend printed material is also seen, but is much less common (Dejerine, 1880). Incomplete examination of the patient, using the ability to read aloud as the sole test of reading, may lead to incorrect labelling of these patients as alexic. By the definition offered at the beginning of this paper it is the comprehension of printed material that is significant so that the patient who comprehends printed material but does not read out loud should not be regarded as alexic while the rare patient who reads aloud without comprehension may be accepted as alexic.

Unilateral Paralexia: Incorrect reading of part of a word or part of a sentence is known as paralexia. This may occur in any alexic patient but of particular interest is the patient who makes paralexic errors only for material falling in one visual field, a unilateral paralexia. The error is either one of omission or substitution, these errors consistently occurring in the part of the word or sentence falling in the hemianopic field when the visual focus centers on the mid portion of the word or sentence. Thus a patient with a right homonymous field defect may read the word 'northwest' as 'north' or 'northern' and the sentence 'He went out to dinner' simply as 'He went out'. The individual with a left field cut may read these as 'west' and 'out to dinner' respectively. As would be anticipated, unilateral paralexia is far more common in patients with left visual-field defects but is also seen occasionally with right hemianopic disturbances. Kinsbourne and Warrington (1962a) reported a detailed study of this disorder, using six cases with right posterior hemisphere

damage secondary to either vascular or neoplastic disease. Each case had presented with distinct left visual neglect and all demonstrated the phenomenon of unilateral paralexia. Each patient could correctly identify individually presented letters and none exhibited any form of aphasia. The reading errors that they made, either omissions or substitutions, occurred exclusively in the left half of space. If, however, the words were presented in a vertical column instead of the traditional horizontal row the full phrase could be correctly and easily read out loud and comprehended. It should be noted that this disturbance does not occur in all patients with a left hemianopia and probably requires inattention in addition to the hemianopia.

This form of misreading may be extremely disabling since the confabulatory responses and omissions of material in the left field may lead to gross distortions of comprehension. These patients are not, however, truly alexic since they do read correctly, both in their good visual field and with vertical presentation. This must then be considered only as a reading disturbance, a pseudoalexia. It may well be similar in basic mechanism to the hemialexia described earlier as resulting from lesions of the splenium of the corpus callosum. It is clear that a necessary component of this disorder is that the patient is unaware of or inattentive to his hemianopia and may either confabulate to complete stimuli not seen in the hemianopic field or omit this area entirely.

Unrecognized developmental dyslexia: It has been our experience on one occasion to examine a patient with a typical Broca's aphasia who had a severe reading disorder. This was not a reading disturbance of the type mentioned earlier during the description of Broca's aphasia, but a true disability in reading. It was only after considerable testing of the patient and questioning of the family that information was obtained revealing that the patient had never been able to read in a normal manner, had attended special schools because of his reading problem and that his reading performance since the onset of aphasia was unchanged from premorbid performance. By the definitions stated in the opening part of this paper this patient would not be considered alexic, but rather a case of developmental dyslexia. It is worth stress since such cases may occasionally present clinically and may be confusing unless this possibility is appreciated.

Mental retardation: Many individuals with significantly limited mental capacity never gain the ability to read. This can be considered as similar to developmental dyslexia but in no sense should be considered as alexia, the individual never having acquired reading ability. Nevertheless, generalized mental retardation is not always obvious to the examiner, especially when dealing with an adult who presents with signs of acute cerebral disorder, and must be considered in the differential diagnosis of reading disturbance. Usually a good history of premorbid intellectual capability will distinguish this disorder.

Confusional state: Inability to read coupled with many other 'mental' symptoms such as disorientation, depressed attention span, hallucinations, apathy, agitation, loss of accepted social manners, agraphia, acalculia, disturbed visual and spatial orientation, etc., is seen not infrequently in both neurologic and psychiatric practice. While the reading disturbance may be profound it is obviously part of the widespread cerebral dysfunction generally known as a confusional state. This state presents a diagnostic problem only when the patient is quietly confused and the other components are overlooked by the examiner. When the confusion clears the reading disturbance will generally disappear along with the other findings. Defective attention (inability to attend to stimuli) is probably the basic factor underlying this reading disturbance which is not a true alexia. It may be difficult in some cases to rule out that confusional disorder and alexia are both present. It should be stressed that isolated alexia is never the result of a confusional state.

Psychogenic disorders: Only on rare occasions will the patient with purely psychogenic dysfunction be considered as alexic. Certainly both severely depressed patients and active or deteriorated schizophrenics may refuse to read upon request, just as they may refuse to carry out other requests but when considered in the context of the entire disease picture the reading disturbance does not stand out. Reports of alexia as a significant hysterical symptom are rare (Teitelbaum, 1941). It would appear that this symptom is too subtle, subjective and lacking in drama for use by the hysterical patient. Thus reading disturbance (pseudoalexia) may well be part of a psychogenic syndrome but it is rarely sufficiently prominent to warrant confusion with true alexia.

IV. ADDITIONAL THEORETICAL CONSIDERATIONS

Hemispheric dominance: In the great majority of cases the pathology of the alexic syndrome is compatible with left-hemisphere dominance although a few cases have been recorded in left handers in whom the pattern of lesions was reversed (Gloning *et al.*, 1966). There is very little evidence which even suggests bilateral representation of reading comprehension. Akelaitis (1941) and Gazzaniga and Sperry (1967) suggested that some of their cases of callosal section showed evidence of bilateral reading ability as discussed earlier in the section on hemialexia. Our experience in a small number of cases of alexia without agraphia has not demonstrated preservation of comprehension in the apparently intact right hemisphere. Thus these patients with right hemianopia and involvement of the splenium but with an intact left visual field not only fail to read aloud but cannot match written words with pictures or objects and fail to carry out written commands with either hand. This problem, in common with other issues of right-hemisphere substitution, deserves further study. It can be stated with some confidence however, that the ability to comprehend written language is dependent upon intactness of the dominant (usually left) hemisphere in the majority of adults and conversely, that the presence of clinical alexia is a strong indication of dominant-hemisphere pathology.

Childhood lesions: We know of no reports of acquired alexia in childhood except in cases demonstrating considerable language loss. Whether this is due to paucity of cases, inadequate examination or because lesions adequate to produce alexia in adult life do not do so in childhood (or would be rapidly compensated) is not known. Descriptions of the clinical pictures, course and pathology of acquired alexia in childhood would be most welcome.

Alexia in the deaf or blind: We do not know whether lesions which produce alexia alone or in combination with other clinical features in the normally hearing and seeing would have the same effects in the congenitally deaf or blind. Thus, if Wernicke's interpretation of alexia as a part of Wernicke's aphasia (cited earlier) is correct, one would expect that a

lesion of Wernicke's area in a congenitally deaf patient who knew only manual language and lip reading might produce no alexia for written material or indeed, for the manual language, since the patient would never have learned these by reference to auditory language and hence would not use the auditory association cortex.

Similarly it would be reasonable to assume that the patient with either acquired or congenital blindness who 'read' Braille would not lose this ability after the usual lesion of alexia without agraphia. What would happen to this patient after the usual lesion for alexia with agraphia? One such case has been observed (Gloning, 1967) and did demonstrate loss of the ability to understand the Braille language as would be anticipated from our earlier discussion of alexia with agraphia.

Relationship to developmental dyslexia: In recent years there has been a resurgence of interest in the problem of the child with exceptional difficulties in learning to read and write. Such individuals have at least a superficial resemblance to the patient with alexia with agraphia in that they may have normal use of spoken language and normal intelligence but are unable to master the written form of language. By our original definition these cases are not considered with the alexias as they have never gained the function of reading but the obvious similarity of symptomatology is at least suggestive of a common factor. To date there are no pathological data available in the developmental dyslexias and any relationship must remain conjectural. It is conceivable that bilateral defective maturation of the angular gyrus could lead to this syndrome but this too is purely speculative.

V. TESTS OF READING

In earlier sections the ability to read aloud and the ability to comprehend written material have been presented as separable functions. It is therefore obvious that they are to be evaluated separately. At the same time, however, it must be recognized that many of the factors which disturb reading capability, may interfere with both types of reading function simultaneously. The following brief outline of test procedures will aid in differentiating varieties of alexia but must always be considered within the entire context of neurologic and aphasic defects.

Reading aloud: In testing this ability the examiner requests that the patient read aloud letters, numbers, words and sentences. In addition to omissions the examiner watches for substitutions, verbal paralexias and omissions or errors restricted to one visual field.

Comprehension of letters: This is tested by having the patient match lower case to capital letters and print to script. If there is failure at this level the patient should be asked to trace the letter with his finder or the letter should be moved slowly in front of the patient (Botez *et al.*, 1964). This test may be made more difficult by cross hatching or other mild disguise of the letter or by tachistoscopic presentation. Number comprehension may be tested by having the patient match a written number with the proper number of objects or fingers.

Comprehension of written words: This is tested by requesting that the patient match a written word with the proper object or body part. Tachistoscopic presentation may be used.

Comprehension of phrases and sentences: This testing can be carried out in several ways. Matching of a short descriptive phrase or sentence with an object or simple yes-no questions can be used. With longer sentences, conditional clauses, unstressed prepositions and absurd statements may be used to probe the quality of comprehension.

Paragraph comprehension: This is tested by offering a factual paragraph followed by questions based on the information in the paragraph. This can challenge both the grasp of specific information and the understanding of the basic ideas put forth in the reading material.

Visual function: Special attention should be given to visual function as it influences reading. This includes tests of visual acuity, visual field determinations of both confrontation and extinction varieties, optokinetic nystagmus, color vision and the ability to identify objects presented visually. Eye movements should be observed and special note taken of the ability to scan a line of print and move to the next line. Complex machinery for recording eye movements during visual scanning has been

described (Garron and Cheifetz, 1967) but is too complicated for use except in research.

Speed of reading: The rapidity with which a patient is able silently to read a passage is obviously of interest but is of limited use in clinical evaluation. There is great individual variation in the speed of reading and a given patient's premorbid ability is rarely known by the examiner. In addition, there are some patients who read painfully slowly but if allowed sufficient time can demonstrate that they have comprehended the material. This may be the only residual of a previous alexia.

Spelling aloud and comprehension of spelled words: One additional evaluation, not truly a test of reading, has proved of distinct value in the study of alexia. This is the determination of the patient's ability to spell aloud and to recognize words spelled aloud by the examiner. Both of these acts will be failed by the patient with alexia with agraphia or with the alexia associated with Wernicke's aphasia. In cases of alexia without agraphia, however, the response to both tests will be at a normal level and individuals with Broca's or conduction types of aphasia will usually recognize spelled words even if they cannot produce the letters to spell a requested word. Failure in both of these tests is suggestive of pathology affecting the dominant temporoparietal area.

The simple test procedures outlined above offer considerable information for differentiation of the varieties of alexia. The ability to read aloud *and* to comprehend letters, words, phrases and longer written language is readily determined and the presence of alexia can be demonstrated. The ability to handle spelled words will aid in differentiating the so-called 'pure' alexia from that produced by dominant temporoparietal disorder. Observation may reveal sources of nonalexic reading disturbances. In the final assessment, however, the exact variety of alexia or nonalexic reading disturbance must be determined by a complete clinical evaluation of which the above-mentioned tests are only one portion.

VI. THERAPY

In the face of the extensive literature available on the alexias including discussions of the clinical state, pathology, physiology, development of

reading skills and breakdown with cerebral disease, there has been surprisingly little specific information concerning therapy for alexia. Most authors are quick to point out that the techniques of remedial reading cannot be employed in alexia without considerable alteration, since the alexic has lost this skill because of cerebral damage whereas the 'slow reader' has yet to accomplish reading. Thus, while remedial reading techniques can be utilized, treatment must be individualized for the alexic patient.

Kinesthetic aid to reading has been emphasized by Goldstein (1948) and many other authors. In essence this consists of having the patient run his finger over the outline of a given letter while teaching him to say the letter name aloud. With continued practice this may become easier for the patient and eventually some patients may develop a kinesthetic sense for eye movements to replace the clumsier and slower use of the hand. This technique, however, has proved unsuccessful in most cases of alexia and at best appears to have limited therapeutic value.

Wepman (1951) emphasizes five steps in therapy for alexia which he admits are borrowed from remedial reading experience. These five steps are:

(1) The determination of residual reading level to provide a starting level for therapy.

(2) The building of a sight vocabulary of simple words, primarily accomplished by intensive drilling.

(3) The phonetic sounding of letters and phrases, again done by intense drilling.

(4) The comprehension of connected written material (sentences and phrases) which is built up by repeated drill on material of interest to the patient.

(5) The continued practice of reading under supervision with occasional testing to ascertain the accuracy of comprehension.

The author points out that the third step may be impossible for individuals with aphasic disorders producing expressive abnormalities and may be deleted. While admitting that alexia may prove difficult to treat, Wepman (1951) suggests that with patience and considerable therapy many patients "can be started on the road to recovery of reading."

For retraining of reading skills Goldstein (1948) suggests starting at a somewhat more elementary level and warns that progress beyond this

level may be difficult or even impossible. He strongly recommends the use of pictures of familiar objects printed together with the first letter of the name of the object (e.g. '*A*' and a picture of an *A*pple). This printed material is combined with verbalization by the therapist until the patient associates the letter and the object and can produce the sound of the letter by saying the known name of the object. Goldstein notes that this technique is frequently used in initiating reading skills in children, but is rapidly dropped as it is both inefficient and unnecessary. In alexia, however, this technique may be necessary for a long period. Another technique advised by Goldstein concerns learning of the alphabet. He advocates dividing the alphabet into groups of five, equating each letter with one of the fingers and drilling sequentially until the alphabet can be recited. Similar drill is then used to equate the spoken letter name with the written letter name. Both Goldstein and Brain (1961) emphasize that some alexic patients perform better when taught immediately to read words without attempting to spell or pronounce letters. This relieves the patient of the arduous task of reaccomplishing phonetic skills, quite impossible for some aphasic patients, but places additional pressure upon the therapist who must beware of paralexic errors.

In summary the therapy of alexia must be individualized, demands arduous labor on the part of both the patient and the therapist plus considerable ingenuity on the part of the therapist and in many cases an end result far below normal levels must be accepted.

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CHAPTER XX

PROBLEMS IN THE ANATOMICAL UNDERSTANDING OF THE APHASIAS

PREFACE

Although it may seem at first to be an odd comparison there is a great similarity between the development of modern genetics and the study of the syndromes resulting from damage to the brain. Although genetics began as a purely 'functional' field, in which one dealt with the inheritance of more or less arbitrarily designated 'characters', the appearance of the idea of chromosomes with genes placed at specific sites created a change in approach. To paraphrase Wernicke who subtitled his 'Symptom-Complex of Aphasia' as 'A Psychological Study on an Anatomical Basis' genetics became a functional study on an anatomic basis. The 'localizationist' approach to genetics has many of the same potential problems as a localizationist approach to the higher functions. Where is the gene for philoprogenitiveness, and does it control the size of a cortical region subserving this characteristic? Clearly it is not reasonable to expect that every nameable feature will have a chromosomal or a cortical localization. Yet this does not mean that certain other aspects of behavior could not be shown to depend critically on a specific gene or a specific site in the nervous system.

Furthermore in order to maintain the theory of localization of genes all sorts of apparently ad hoc explanations had to be advanced, e.g., crossing-over, repression, etc. Yet the theory has stood up because the ad hoc explanations have been of such a nature that they could readily be subject to experimental verification.

The anatomical approach to the higher functions has similarly stood up to many of the same diffuculties. The definition of what is localized remains a problem. Furthermore the fact that a lesion which usually produces an extensive clear-cut syndrome will in other patients lead to a more restricted syndrome or to no difficulties at all can be handled by theories similar in many ways to those which account for the lack of genetic disorder in patients who lack an apparently critical gene. In

other words, just as in some cases a second gene at another site may produce the same enzyme as a deleted gene, in some patients other areas or alternative routes can account for the lack of expected deficits. What saves these explanations from the easy accusation of being ad hoc, and what makes them superior to an invocation of plasticity, is that they are potentially testable.

Over the several years since this paper was written the fact that anatomical approaches to the higher functions have again become acceptable has made it possible to turn with greater care not to the things that the theory explains easily but exactly to the areas where the theory seems to fall short. My own guess is that these will represent not a threat to the anatomical approach but will instead open the way for new investigations which will refine our anatomical notions. The paper presented here was, by my current standards, an early attempt. The publication of, for example, Luria's data on recovery from aphasia after penetrating brain wounds, would, for example, have to be considered more extensively in any contemporary discussion.

PROBLEMS IN THE ANATOMICAL UNDERSTANDING OF THE APHASIAS*

1969

In the classical period that ended approximately with World War I, the approach to the aphasias and other disturbances of the so-called higher functions of the nervous system relied heavily on anatomical knowledge. Broca's (1861) paper, which revealed for the first time that localized lesions of the nervous system could lead to disorders of language, stimulated an enormous interest in the founders of the infant specialty of clinical neurology. Wernicke's (1874) paper published thirteen years later opened the period of the great investigations that continued for another half-century. Stimulated by Meynert's sketch of the major outlines of the cortical connections, he attempted to account for the syndromes of aphasia. A major portion of the advances in the field were made by either his assistants or students in Breslau, or by others following his theoretical approaches (Geschwind, 1966, 1967b). This approach was the only one extensively capable of generating predictions. The other two approaches – i.e., the holistic approach and the mosaicist approach – each had protagonists who made important contributions but neither of these approaches ever matched the successful predictive capacity of the connectionist approach of Wernicke. Although it is commonly believed that the teachings of Wernicke's school were seriously challenged, particularly by the holists, none of the opponents could advance major evidence which threatened the general outlines of aphasia as developed by the Breslau school. Thus Kurt Goldstein (1917) pointed out that his disagreements with the classical localizationist teachings extended only to finer aspects of localization too subtle to be dealt with by current anatomical techniques. As has been pointed out elsewhere (Geschwind, 1964), Goldstein not only accepted the major of Wernicke but made major contributions in the same theoretical tradition. Head (1926) totally rejected this approach, but in his own work ended up with a series of localizations that agreed with those of his classical predecessors.

Although the connectionist approach to disturbances of the higher functions of the nervous system has been highly successful, it should by

no means be thought that it has led to more than an initial understanding of the problems posed by these disorders. Even among those generally committed to this approach, there has always been heated controversy, and the supporters of the opposing mosaicist and holistic schools repeatedly pointed out other areas in which the classical approaches led to predictions at variance with the observed data or phenomena and which they were incapable of handling. It is the purpose of this paper to stress not the successes of the classical anatomical interpretation but rather the areas in which it encounters difficulties. We will begin with a brief outline of the current state of theory and then devote most of our discussion to these problem areas that constitute some of the chief challenges for future research.

I. THE ANATOMICAL BASIS OF THE HIGHER FUNCTIONS

Let us for a moment omit any discussion of the problem of cerebral dominance and consider that the functions we are concerned with are present to a significant degree only in the left hemisphere. We will summarize broadly the functions of certain areas (Figure 1). The angular gyrus region (or more correctly, the region of the temporo-parieto-occipital junction) may be thought of as containing the rules for associating stimuli in two modalities, e.g., visual and auditory. Thus a visual stimulus can evoke an auditory association by means of the pathway: visual cortex – visual association cortex – angular gyrus – auditory association cortex. The auditory association cortex shown here is the classical Wernicke's area. Broca's area is a region of motor association cortex lying anterior to the face region of the classical motor cortex. It may be thought of as containing the learned rules for translating a particular heard sound pattern into a motor sequence.

In the light of the above scheme let us consider how we name a seen object. A stimulus reaches the visual cortex, goes to the visual association cortex, then to the angular gyrus. This in turn arouses the name in Wernicke's area. From here the impulse is transferred over the arcuate fasciculus to Broca's area where the muscular pattern corresponding to the sound is aroused. From here the impulse reaches the motor cortex and is then conveyed downward.

This simple diagram immediately permits us to make certain predic-

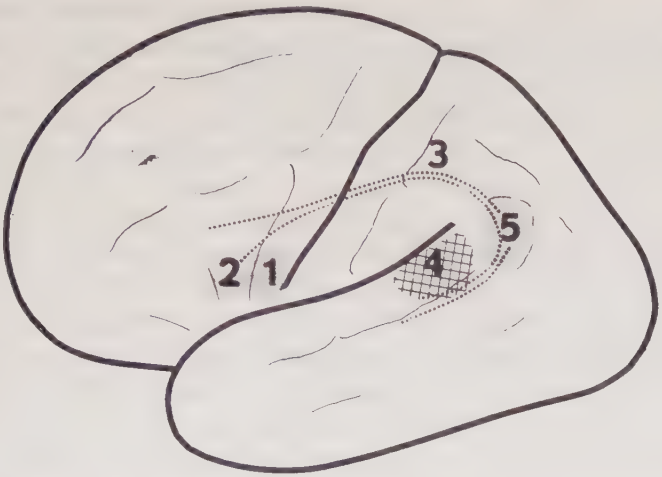

Fig. 1. Lateral surface of left hemisphere. (1) Face area of motor cortex; (2) Broca's area; (3) lesion involving arcuate fasiculus (dotted lines); (4) Wernicke's area; (5) Angular gyrus.

tions. Assume a lesion of Wernicke's area. A word may reach the auditory cortex but can arouse no response in Wernicke's area and thus fails to arouse associations elsewhere in the brain. Hence it is not understood, nor can it be repeated. The patient will not be able to describe his environment since a stimulus from visual cortex will not eventually reach Broca's area. The patient fails to understand written language since the seen words can no longer arouse their auditory forms. A contrasting lesion in Broca's area produces a different pattern. Speech is abnormal but comprehension of spoken and written language is preserved. A lesion in the arcuate fasciculus also leads to abnormality in speech by breaking the pathway from Wernicke's to Broca's area but comprehension of spoken and written language is retained. The disturbance produced by a lesion in this location is called conduction aphasia.

All of the lesions mentioned above lead to abnormal speech, but the disturbances vary with the localization. Thus in the lesion of Broca's area little speech is produced and what is produced is halting and dysarthric. In the other two forms mentioned above, there is often copious production of abnormal speech. It is clear that the integrity of Broca's area is necessary for the production of this type of fluent aphasia. It

seems reasonable to hypothesize that this fluent abnormality represents the running on of the relatively isolated Broca's area. Some support for this view comes from the fact that in children fluent aphasias either are never seen or are extremely rare. This would appear to be the result of the fact that Broca's area has not had the overlearning necessary to permit its running on in isolation.

Other syndromes can also be predicted from this model. Consider a lesion that destroys the left auditory radiation or cortex and that also destroys the callosal fibers from the opposite auditory region. Wernicke's area, although intact, can now receive no auditory stimulation. The patient does not understand spoken language although he speaks normally since Wernicke's area is intact. An analogous situation is seen when a lesion destroys the left visual cortex and the splenium of the corpus callosum. The patient can see words in the left visual field, but these are not capable of reaching the speech areas and the patient can no longer understand written language, although he speaks and writes correctly.

A similar analysis enables us to understand a related group of disturbances, i.e., the apraxias. Consider how a patient carries out motor responses to auditory command. It is necessary to use the pathway from Wernicke's area to the left motor association cortex and from there to the motor cortex. To carry out auditory commands with the left hand, it is necessary to go by way of Wernicke's area, left motor association cortex, then over the corpus callosum to the right motor association cortex and the right motor cortex.

A lesion of the corpus callosum thus causes the patient to be incapable of carrying out motor commands with the left hand while he is still capable of performing them with the right. A lesion of the left motor association cortex will have the same effect, although the praxic difficulty in the right hand is often masked by a right hemiplegia. A lesion in the arcuate fasciculus will lead to a bilateral difficulty in carrying out verbal commands.

II. DIFFICULTIES IN ANATOMICAL INTERPRETATION

We may divide our difficulties of interpretation into three categories.

(1) There exist certain regular features of the syndromes listed above

that do not appear to be readily explained by the anatomical model.

(2) There exist certain syndromes whose existence is not predicted by the model.

(3) There exist certain cases where the expected syndromes do not appear despite an apparently adequate lesion.

The occurrence of difficulties of any of the above types has led many authors to dismiss all localizationist approaches as incorrect, but without offering an adequate substitute. Despite its shortcomings, an anatomical approach most closely meets the criteria of efficiency in explaining the known data, efficiency in predicting new phenomena, or in design of important experiments and susceptibility to refinements that can be checked by observation or experiment. In some instances the discrepancies between theory and observation are only apparent. Thus Wernicke himself discussed the syndrome to be expected from a lesion disconnecting Wernicke's area from Broca's area. His predictions were apparently not borne out, but subsequent studies showed that there were two reasons for this: (1) Wernicke had incorrectly assumed that the pathway between these two regions traversed the insula; (2) he had incorrectly interpreted his own diagram in determining what the effects of such a lesion should be.

Let us now consider some of the specific difficulties that arise. The objection might be raised that the expressive disturbance should be the same in the case of Wernicke's, conduction, and Broca's aphasias. As we have already noted, this is not the case since the two former varieties lead frequently to fluent aphasias while Broca's aphasia is nonfluent. This is probably accounted for by the hypothesis mentioned earlier that the fluent aphasias represent situations in which Broca's area runs on by itself. However, certain features of these syndromes are not predicted by the approach given above, although they are not inconsistent with it.

Thus the linguistic production is different in the fluent and the nonfluent cases. The nonfluent cases tend to have relatively less trouble in naming but marked difficulty in producing sentences, while the fluent group may produce 'empty' sentences that are well constructed but lacking specific words and have great difficulty in naming. This possibility suggests that the anterior speech region is involved in some special way in the grammatical structuring of speech, but the mechanism of this is not at all clear at this time. Why should naming often be relatively well

preserved in the Broca's aphasia? There are at least two alternative explanations available to us. Let us assume that in the posterior speech regions we have a system for finding the word appropriate to a given physical stimulus. A partial lesion will result in either failure to find the word or selection of the wrong 'bin' that will contain an incorrect word. Assuming this posterior region to be intact, then the correct auditory form of the word is aroused in Wernicke's area and is then transferred forward. What reaches Broca's area is thus the correct 'sound-pattern' which must arouse the appropriate muscular patterns. A partial lesion in this region will result in inaccuracy of these patterns but the word may be correct. It should be stressed, however, that the only effect of Broca's area lesion is not an inaccurate sound pattern since the grammatical structure is also distorted for reasons that are not clear and that are certainly not clarified by our present anatomical knowledge.

An alternative explanation of the relative preservation of naming in Broca's aphasics is that the right hemisphere can carry out naming tasks. This, however, probably cannot explain the fact that this preservation of naming is relatively common with left anterior lesions but not with posterior ones. A difference in the form of coding anteriorly and posteriorly as outlined above seems more likely. Hence, right hemisphere substitution, while possible in certain situations (as we will discuss below), is at least not a general explanation for the preservation of naming in Broca's aphasics.

If the model given is correct then why does not a lesion of the left internal capsule lead to some permanent abnormality of speech, even if it is only dysarthria or mutism? Yet the evidence seems to point to good recovery of speech after left purely capsular lesions. The reason for this is the fact that an alternative pathway exists. If Broca's area is intact as well as the callosal fibers from it to the corresponding region in the right hemisphere, then the impulse can cross to the right side and eventually the motor impulses may be transmitted down the right internal capsule. Since each Rolandic face area controls nearly all of the articulatory system bilaterally, the speech will be normal. This explanation is supported by the case of Bonhoeffer (1914) who was thought to have a Broca's aphasia in life but who at postmortem had a callosal lesion as well as a left capsular lesion. It is also supported by the fact the Broca's aphasia can be produced by subcortical lesions that simultaneously

involve the callosal fibers from Broca's area and the descending fibers from the left face area.

A more serious problem is that raised by disturbances of writing. How does one write the name of a seen object? Some of the classical authors were inclined to feel that the sight of the object could directly arouse the written production, just as they felt that the production of the spoken name could take place over a direct pathway from the visual region to Broca's area. But what makes this supposition unlikely is that writing is *invariably* abnormal in patients with the speech *pattern* of Wernicke's aphasia,[1] while one might expect it to be spared at least occasionally if this were the case. Hence it appears that to write the word, the spoken form must be aroused first. But what is the next step? It seems possible that it is necessary to go from Wernicke's area back to the angular gyrus again since one can get an isolated alexia and agraphia from angular gyrus lesions, an agraphia that involves even the writing of dictated words.

Even this complicated arrangement leads to difficulties. Is there a separate pathway forward from the angular gyrus to the motor region? If this is the case, then how do we account for the fact that patients with Broca's aphasia have, in my experience, invariably manifested a writing disturbance of generally comparable degree to their speech disturbance.[2] It would seem reasonable, at first glance, that writing should not depend on Broca's area, which appears to be association cortex for the face region of the Rolandic cortex. Why then should writing be affected in these cases? The assumption that somehow the 'heard word' passes from Wernicke's area to Broca's area, there to be turned into written as well as spoken form, does not appear to fit in with the already mentioned fact that pure alexia with agraphia can be produced by angular gyrus lesions. One can make conjectures that are as yet not verified. If the pathway from the angular gyrus runs forward and mixes in the lower parietal lobe with the fibers from Wernicke's area, then one would expect agraphia with conduction aphasia, which is indeed always the case. One could further assume that the face and hand area overlap considerably in the premotor region so that any lesion large enough to produce aphasia will also produce agraphia. Yet this overlap cannot be complete since while pure Broca's aphasias are not seen, it seems to be the case that an occasional patient does develop agraphia without

disturbance of speech from a lesion in front of the hand area of the cortex.

It can be seen that we have a reasonable knowledge of which lesions produce writing disturbances, whether in isolation or in association with other difficulties, but we have only a poor notion of the possible mechanisms.

Another anatomical mystery is afforded by the transient aphasias that come from lesions of the supplementary motor cortex. This region is not shown in Figure 1. It is, however, striking that this is the only cortical region outside of the classical speech areas from which aphasic phenomena are obtained on stimulation (Penfield and Roberts, 1959). My colleague, Dr. Deepak Pandya (personal communication), has shown in his anatomical studies in the monkey that the face portion of the supplementary motor area is clearly connected with the face portion of the classical motor cortex.

The first suggestion of the importance of the parasagittal region for speech is found in the work of Schwab and Foerster (Schwab, 1927), who found that 14 out of 21 patients with parasagittal cortical excisions, well remote from Broca's area, developed an aphasia beginning about the third postoperative day and lasting for two weeks or even longer. Transient speech disturbances from lesions in this area are mentioned by several authors (see Penfield and Roberts, 1959). The fact that these aphasias are transient has probably led to a neglect of this region. What role this area plays in speech still remains to be determined.

One of the hardest syndromes to account for, oddly enough, is classical anomic aphasia. One would be tempted to say that a lesion in the angular gyrus produces a visual-auditory disconnection and hence a disturbance of naming. But this explanation affords great difficulties, which have been discussed elsewhere in some detail (Geschwind, 1967a). The first problem is that classical anomic aphasia is predominantly a one-way disorder, i.e., the patient finds it difficult to find the name of a seen object but usually recognizes the name readily when it is offered by the examiner. This contrasts with the color-naming disturbance seen in many cases of pure alexia without agraphia, which appears to depend clearly on visual-auditory disconnection. Here the patient can neither give the correct name of a seen color nor recognize the name when it is offered. He clearly suffers from the inability to match the heard word to

the visual stimulus, but this match fails in both directions, as one would expect in an audiovisual disconnection.

A clue to the problem, although not a solution, is given by condideration of another fact. It is frequently stated that lesions of the second temporal gyrus are involved in anomic aphasia, while other authors stress the angular gyrus. Indeed, these are not necessarily conflicting views, since the second temporal gyrus is in fact continuous with the angular gyrus. Like the angular gyrus of man, the second temporal gyrus is very late in its maturation (Flechsig, 1901). It is conceivable that we may have simplified too much in assuming that there was a simple step from angular gyrus to Wernicke's area for arousing the name of a seen object. We frequently must find a 'name' in speech, using several cues, each one inadequate by itself. It is like the question: "What is an animal, found in India, savage, with stripes?" The word 'tiger' is easily found, yet none of the clues alone is adequate, and omitting even one might still leave difficulties. Presumably the process of word-finding in spontaneous speech may well be similar. All sorts of partial information, sometimes even in different modalities, must come in and be used to find the right word, Conversely, given the word, finding the attributes is easy. It would appear to me reasonable that anomia is not the result of auditory-visual disconnection (which is probably not present in the anomic) but rather a disturbance of a well-organized 'filing system' that perhaps is present in the angular gyrus and second temporal gyrus. But we need much further information to support this view. Isolated vascular lesions of the second temporal gyrus are remarkably uncommon and hence clinical information is lacking. Unfortunately we know very little of the connections of this region.

Even in the purer forms of aphasic disturbance we run into difficulties. Consider the syndrome of pure alexia without agraphia. The lesion involves the left visual cortex plus the splenium of the corpus callosum. It seems clear that this lesion isolates the right visual cortex from the speech area and the seen word cannot arouse the auditory form. How then do we account for the ability of the patient to name objects that must surely also require a connection from right visual region to the speech area? This question is dealt with elsewhere in some detail (Geschwind, 1965) and will be discussed briefly here. One possible explanation is that object-naming is preserved by means of arousal of

tactile associations in the right hemisphere which can then get across the callosum in its intact portions. Consistent with this explanation is the fact that in many of these patients the ability to name colors is impaired along with the disturbance of reading. Colors, unlike objects, will not have distinctive tactile associations and hence may well be disturbed along with reading.

Another problem not accounted for adequately by this model is the curious syndrome of aphemia. A typical case is that of a patient who suddenly becomes mute in association with a right hemiplegia. The patient, although mute, can write. Pseudobulbar mutism appears to be excluded by the absence of other pseudobulbar signs. The hemiplegia may clear almost completely. As soon as the patient has some speech, it is usually evident that his language production is normal although his speech is slow and effortful. He may recover to the point where speech is produced readily enough for the purposes of ordinary life, but the patient remains markedly dysprosodic. The designation of this syndrome as 'subcortical motor aphasia' appears to be inappropriate, since it seems highly unlikely that a lesion cutting off Broca's area from the Rolandic face area could frequently do so without a permanent hemiplegia. This syndrome for the time being remains a mystery.

The usual ability of the aphasic patient to sing on key correctly is particularly dramatic in the case of the classical Broca's aphasic whose spontaneous speech output is so limited and yet who generally sings melodies well. Even more striking is the occasional situation when words as well as music are produced in this situation, this being sometimes the only circumstance in which the patient produces any words. The interpretation that 'music is in the right hemisphere', although possibly correct, is not very helpful if one is attempting to understand mechanisms. If Broca's area is in some sense a storehouse of motor patterns corresponding to auditory patterns, then why should music not be affected? We would have to assume that the nervous system separates the musical auditory stimuli by some set of criteria into the right hemisphere. This is certainly possible but it would be interesting to know what set of criteria would be used by the nervous system. In any case this problem remains unsolved.

Another difficult problem is posed by the anatomical model given above. How do we carry out certain commands such as "Pick up the

ball?" A connection from auditory region to motor region alone would not suffice for this task since visual information is also necessary. This implies that perhaps the auditory and the visual information are 'mixed' somewhere before being relayed to the motor region. This would of necessity involve some modification of our ideas of the apraxias. It would suggest that, rather than a direct auditory-motor pathway being involved, we use a pathway with a synapse in it. On the basis of anatomical studies in primates carried out by Pandya (personal communication), it seems reasonable that this region lies in the inferior parietal lobule, but much more investigation will be required to establish this view.

III. LACK OF EXPECTED EFFECTS OF LESIONS

Let us now turn to another problem. As we have pointed out, we lack the ability to account for some of the common features of syndromes whose anatomical basis appears to be well established. Nor can we yet account for some syndromes such as aphemia that are not accounted for on the basis of the anatomical structure outlined above. We now turn to a different problem. How do we account for the situations where an apparently adequate lesion fails to produce the expected syndrome? Thus Kleist (1962) describes a patient with a destructive lesion that would have been expected to produce a Wernicke's aphasia, yet it produced no comprehension disturbance, although repetition was impaired. The problem is, therefore, why are some of the expected elements lacking or, indeed, why in some instances is there a total lack of the expected syndrome?

It should be pointed out that this is not the argument against localization that it is sometimes purported to be. The problem posed here is why certain lesions are necessary but not sufficient conditions for the existence of certain syndromes. Yet any reasonable theory must eventually account for the exceptions in a testable manner. We will consider here some of the possible explanations for this type of situation. For some of these good evidence exists, while others are speculative but subject to eventual test.

Of course one of the obvious reasons for discrepancy is inadequate examination. Thus patients are sometimes asserted to have difficulty reading as a result of Broca's area lesions (Nielsen, 1939). While it is

certainly possible that such cases exist, the evidence is by no means clear, and it is quite evident that some of the cases so described have had difficulty in reading aloud with preserved comprehension of reading. Another common error is to diagnose difficulty in comprehension in patients who fail to carry out commands on the basis of apractic disturbances. This error is a frequent one, since most clinical tests for examining comprehension employ only the ability to carry out commands.

Another reason for apparent discrepancies is reliance on cases of cerebral tumor. As is well known, there are often marked discrepancies between the site of a tumor and its clinical symptomatology; these may often be the result of distant effects of distortion of the brain, traction on blood vessels, and obstructions of the ventricular system. Similarly, one can expect discrepancies in cases of cerebral infarction who die early in their course, since edema around the area of destruction may mislead one as to the effects of the destruction itself.

Another problem that may lead to apparent discrepancies is the transient syndromes such as those mentioned earlier from the supplementary motor area. There are probably other such syndromes that have been overlooked because of their transiency. Thus it is our belief that frontal lesions in man commonly lead to transient but not permanent unilateral inattention syndromes just as they do in animals (Welch and Stuteville, 1958).

Let us now turn from these cases of what we may call apparent discrepancy to cases of true discrepancy.

One of the oldest theories is that the way a task is learned will affect its performance. Thus one can theoretically conceive of a congenitally deaf person who has learned to read but has never learned spoken language. Since he has not learned to read by translating the written words into auditory form, he should not lose reading as a normal person would from a Wernicke's area lesion. Could one apply this type of reasoning to normals? Wernicke (1874) himself argued that one should see different aphasic pictures in the highly educated. One can imagine that eventually one might 'free' himself from translating written language into auditory form and thus not lose reading with a Wernicke's area lesion. Similarly many older authors argued that the poorly educated who read aloud could become alexic with a Broca's area lesion. Unfortunately no one has yet published an adequate study of alexia from a Broca's area lesion.

All of the above suppositions are possible but as yet unproven. At least one hypothesis of this type is probably incorrect. Thus some authors have argued that unilateral cerebral dominance for speech is the *secondary* result of having learned to write with one hand, usually the right. On the basis of this, it has been argued that speech would be less localized in the illiterate and hence their aphasias would be milder. Many lines of evidence argue against these hypotheses. The overwhelming predominance of left hemisphere lesions in the aphasias appears to have been as marked in France and Germany of the late 1800's, when illiteracy was common, as it is today. Despite the fact that nearly all left-handers of the late nineteenth century on the Continent were taught to write with the right hand, aphasias in association with left hemiplegias were frequently observed in such patients. At least half of those who today do write with the left hand become aphasic with right hemiplegias. There is further evidence against this thesis from callosal lesions in left-handers. It seems unlikely that the use of one hand for writing controls cerebral dominance for speech.

A second mechanism for incomplete or absent syndromes is duplication of function in the right hemisphere, either because the function was always duplicated or because of relearning. The evidence for duplication of function in varying degrees is quite strong. Thus the child who becomes aphasic frequently recovers very good speech within a few months. The period is clearly too short to permit relearning. Hence, language must have been in the right hemisphere before the lesion. Why is recovery not always immediate? It seems most likely that the right hemisphere is normally inhibited and that some period is required to release this inhibition. There are many analogs to this even in normal life. The 'savings' technique, one of the classic methods of study of memory, is frequently used to show that there is some preservation of memory despite even gross failures in recall.

The conclusion that the child in many instances does not relearn with the right hemisphere but rather must somehow bring to the surface learning already present has some important implications. It necessitates recasting of some conventional ideas of cerebral dominance. We are often accustomed to thinking of dominance as the predominant *possession* by one hemisphere of a certain skill. Perhaps a more reasonable view is that learning takes place, at least in childhood, on both sides in roughly

equal degrees, but that retrieval is readily effected only from one side.

The second important implication is that it is conceivable that, if language was once present on both sides, it is always present on both sides but that in the adult the retrieval problem is not fully overcome. Certainly if retrieval often takes a few months in the child, the period might extend to years and in some cases retrieval might never be achieved in the adult. It is my belief that the major hope for the eventual effective rehabilitation of the type of aphasic for whom we can do so little now lies in the possibility that language is present but inaccessible in the minor hemisphere.

Similar situations to those seen in childhood do exist in varying degrees in adult life. The left-handed patient of Dejerine and André-Thomas (1912) became aphasic with a left hemiplegia and then had a slow recovery. By the time of her death about five years later, speech was nearly normal. Relearning could probably be excluded by the patient's ability to deal with discussions of matters that she would hardly have dealt with during her illness, and by the high character of the performance. At post-mortem the patient had nearly total destruction of the right hemisphere so that she must have been using her left hemisphere for recovery of speech. Cases similar to this one, although not perhaps so dramatic, are seen by everyone who works with aphasic patients. In ordinary practice, it is not unusual to see a patient who a year after the onset of aphasia is still severely impaired, yet who two years later shows a marked improvement. Lesser degrees of obvious right hemisphere substitution are also seen. Thus the patient studied by Smith and Burklund (Smith, 1966) who underwent a total left hemispherectomy for a glioma was severely aphasic, but he did have some speech and this was obviously coming from the right hemisphere. This certainly must suggest that, in other patients with severe aphasias, some of the retained speech comes from the right hemisphere. It seems likely that there is a great deal of variation in the extent of right hemisphere substitution.

The thesis of right hemisphere substitution need not be regarded as simply an *ad hoc* explanation, since there are means for studying this problem. Wada tests on the right carotid artery in aphasics at different stages of recovery would help to show how much speech was in the right hemisphere. We have, however, been reluctant to carry out arterial punctures for this purpose alone. Even this technique could not demon-

strate the full potential of the right hemisphere but only its actual performance at any given moment.

A third possibility to account for incomplete or absent syndromes is not a shift to another cortical region but rather the use of alternative pathways to perform the function. There are two possibilities here. One is that of circumventing a disconnecting lesion by means of other pathways. An example is seen in animal work. Thus Black and Myers (1964) showed that, with lesions of the splenium of the corpus callosum in chimpanzees, a minority of the animals could effect transfer of visual learning between the hemispheres as long as anterior commissure was intact. This makes sense anatomically since the visual association areas (which are linked to their opposite numbers by means of the splenium) project to the lateral and basal temporal lobe which connects to the opposite side via the anterior commissure. We see similar situations in man. Thus as Bonhoeffer (1914) pointed out, a left capsular lesion does not produce aphasia because one can use the connections from the left Broca's area to its analog on the right. Bonhoeffer described a patient who became aphasic with the combination of a left capsular lesion plus a callosal lesion. It seems likely that the commissural pathway from Broca's area is commonly used in man when there is a capsular lesion. There may well be other situations where some humans can make use of a commissural connection (like the minority of chimpanzees who use the anterior commissure when the splenium is cut) that others fail to use. This will clearly lead to variation in clinical pictures with the same lesion. Notice that this is not the same as substitution by the right side. Substitution can conceivably occur when the left hemisphere is totally destroyed. In the type of situation we are talking about here connections from the intact portions of the left side are required. Substitution implies that a cortical region on the right can carry out the function. The type of compensation we are discussing here may merely mean that the right side is being run passively from the left. Obviously combinations of the two mechanisms could occur.

Still another type of mechanism involving alternative pathways must be considered. It is clear from anatomical studies that large motor outflows exist from regions other than the frontal lobe. Kuypers has laid special emphasis on this fact (Lawrence and Kuypers, 1965). These directly descending motor systems seem to arise primarily from the sensory

association cortices and descend subcortically, in particular to the collicular or pretectal regions and to the cerebellum via the pontine nuclei (Jansen and Brodal, 1954). These pathways are probably not the complete equivalent of the pyramidal pathway. They are, however, capable of the delicate control of eye movements. The eye movements are in fact probably all controlled by this type of system, since there seems to be no representation of eye movement in the pyramidal system. On the other hand, these systems appear to be capable of less finesse in fine distal limb movements and seem to control mostly axial musclature. These nonpyramidal systems are probably much older phylogenetically than the pyramidal system.

There is a certain amount of evidence to show that at least in primates the direct descending pathways from the sensory association cortices may be less important in the normal animal than the transcortical pathway via the frontal lobe. With removal of the transcortical pathway, however, the animal can compensate to a great extent by means of the direct descending systems but the compensation may not be perfect. Thus Glickstein, Arora, and Sperry (1963) showed that for delayed response tasks it is still necessary to use the transcortical pathway. The important point, however, is that such direct descending pathways from sensory association cortices can often produce a significant degree of compensation in the primate. We must ask how great a role they play in man.

Some activities in man probably use directly descending pathways normally. Thus, even after destruction of the corpus callosum, walking is carried out normally. It seems unlikely that this activity need be coordinated via the corpus callosum but probably depends on these directly descending pathways. The better preservation of walking and other whole-body movements in many patients with apraxias resulting from destruction of cortico-cortical connections is compatible with this view.

There is other evidence for the participation of direct descending pathways in man. They may well account for the return of some movements of the left arm to verbal command in patients with callosal lesions. Thus we have had the opportunity, through the kindness of Drs. Bogen, Gazzaniga, and Sperry, to examine the first patient who underwent callosal section in Los Angeles. He failed to carry out many commands with the left hand but did succeed in a minority of instances. How can we account for this partial performance? Direct ipsilateral pathways from

the left hemisphere appear to be the most likely explanation. Such ipsilateral pathways are most likely to be from the 'medial' motor system of Kuypers (Lawrence and Kuypers, 1965) rather than from the 'lateral' system whose connections are more strongly contralateral. Further evidence for the participation of these medial systems in this patient is that he carried out the commands in a different way in the left and right arms. With the left arm he tended to lose the finer distal components of movements but not with the right, findings compatible with the notion that the 'medial' system was playing a greater role in the performances of the left hand. The same situation of partial carrying out of the commands with loss of the finer distal components is seen not only in lesions of the callosum proper but also in the apraxias of the left arm accompanying Broca's aphasia. It seems reasonable that here, too, the participation of direct descending 'medial' motor systems can be invoked.

Could such direct descending pathways be adequate for the restoration of motor speech in the Broca's aphasic? I should think that this could give at best a partial restoration since the articulatory pattern of normal speech is so refined that I doubt the capacity of the medial system to achieve it. This problem, however, deserves further study. Conceivably, the relative and often dramatic preservation of naming in the Broca's aphasic might depend on such direct descending pathways from the posterior speech regions. Whether such pathways can maintain fully normal speech is an open question.

Let us now turn to the last of the mechanisms of the preservation of performance in the face of lesions which seem adequate to prevent it. This is the mechanism of 'external' signaling, i.e., where one portion of the patient's nervous system stimulates visually or mechanically another part of the nervous system to which it does not have access by means of nervous connections. Let me cite two examples. A patient with facial apraxia, on being asked how he would blow out a match, may make the motion of striking a match and then carry his hand up to his face. He may then carry out the blowing movement. If he is prevented from using the arm, he will not carry out the blowing movement. It is clear that the command has been understood and that the patient is capable of carrying it out, but he can do so only by means of stimulating visually a portion of the nervous system that cannot be reached by internal connections from the left auditory region. Similarly, a patient with a callosal lesion,

on being asked to salute with the left hand, may first carry out the movement with the right hand and then with the left. If the right hand is restrained, he is incapable of the action. Again it seems likely that the left hemisphere, which understands language, is signaling the 'aphasic' right hemisphere nonverbally.

This concludes our discussion of the problems involved in anatomical interpretations of the higher functions of the nervous system. It has by no means exhausted them and only further anatomical and physiological knowledge, coupled with clinical observation and testing, will serve to unravel the difficulties presented here.

NOTES

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[1] I have never seen a patient with grossly fluent and paraphasic speech who produced normal written language, although the orthography may be quite normal, nor have I seen a convincing report of such a case.

[2] There are occasional reports in the literature of 'pure motor aphasias' involving only speech and not writing. My own experience, however, has been that patients who are mute but write normally manifest normal language production as soon as they have any speech although they may be extremely dysarthric. Patients with the clear-cut linguistic pattern of Broca's aphasia in speech but with normal writing must be extremely rare, if they exist at all. Certainly some degree of discrepancy between written and verbal production is common but is not dramatic.

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CHAPTER XXI

THE ORGANIZATION OF LANGUAGE AND THE BRAIN*

Language Disorders after Brain Damage Help in Elucidating the Neutral Basis of Verbal Behavior

1970

ABSTRACT. The study of the organization of the brain for language has been based of necessity on investigations in man. The bulk of our information in this area has come from careful studies of patients suffering from isolated damage as a result of vascular disease, whose brains have, after death, been subjected to careful anatomical examininations. Disorders of language resulting from brain damage, almost always on the left side, are called aphasias. Carl Wernicke, nearly 100 years ago, described the linguistic differences between aphasias resulting from damage in different anatomical locations and outlined a theory of the organization of language in the brain. Not only have Wernicke's localizations stood up under repeated examinations but his theory has been the only one which has permitted the prediction of new phenomena, or has been able to account for new observations. Several remarkable disorders, such as isolated disturbances of reading and the symptomatology of the corpus callosum, are examples of the explanatory power of this theory.

The phenomenon of cerebral dominance – that is, the predominant importance of one side of the brain for a class of learned behavior – occurs, as far as we know, in no mammal other than man. The dominance of the left side of the brain for speech is the most striking example of this phenomenon. Contrary to generally accepted views, there is a striking anatomical asymmetry between the temporal speech region on the left side and the corresponding region of the right hemisphere.

Many problems relating to the functions of the nervous system can effectively be studied by investigation in animals, which permits controlled and repeatable experiments on large groups of subjects. When we come, however, to consider the relationship of the brain to language, we must recognize that our knowledge is based entirely on findings in man. Some authors would even argue that language is exclusively a human attribute, so that no experiments on animals could ever be relevant. Although I believe that forerunners of language do exist in lower forms[1], the direct contributions to this area of experimentation on the brains of animals still lie in the future.

I. BRAIN LESIONS IN MAN

Information in this area has come from several sources. Cases of brain tumor are of limited value, since tumors distort the brain and produce

effects at a distance. Cases of penetrating brain wounds[2] have been of considerable use but are not the best source of anatomical data, since postmortem information is usually lacking. Analysis of the sites at which the skull was penetrated is of use statistically, but, because of variations in the paths taken by missiles, cannot provide precise data concerning the location of lesions producing language disorders. Stimulation during surgery[3] has been another most important source of information but, because of limitation of time at operation and the accessibility of only certain structures, has not covered the full range of phenomena observed clinically.

The elegant studies of Milner and her co-workers on patients undergoing excision of cortical regions for epilepsy represent the largest corpus of truly experimental studies of the higher brain functions in man.[4] They are limited, however, with respect to the range of phenomena observed. Furthermore, since most of these patients were undergoing removal of areas of brain which had been the site of epileptic discharges since childhood, there is reason to believe that the effects seen after surgery may not represent the full range of phenomena seen after damage to the adult brain. The Wada test[5] in which sodium amytal is injected into one carotid atery, has been a major source of knowledge concerning the lateralization of language functions in the brain.

Although important information has been obtained by the above methods, it is still true that the bulk of our knowledge concerning the relationship of the brain to language has been derived from the study of adults in whom delimited areas of brain have been damaged as the result of occlusion of blood vessels, who have been studied carefully over long periods, and whose brains have been subjected to careful postmortem examination. Although fully suitable cases of this type are not common, the experience of nearly 100 years of study has built up a large body of reliable knowledge.

II. APHASIC DISORDERS

The generic term *aphasia* is used to describe the disorders of language resulting from damage to the brain. Early in the history of the study of aphasia the distinction between language and speech was stressed. In disorders of speech the verbal output was impaired because of weakness

or incoordination of the muscles of articulation. The criterion of a disorder of language was that the verbal output be *linguistically* incorrect. The muscles of articulation might be used normally in nonlinguistic activities. Similarly, in aphasic disorders of comprehension the patient might lose the ability to comprehend spoken or written language and yet show normal hearing or vision when tested nonverbally. Furthermore, these disorders could occur without impairment of other intellectual abilities. The aphasias were thus the first demonstrations of the fact that selective damage to the brain could affect one class of learned behavior while sparing other classes, and thus gave origin to the field of study of brain-behavior relationships. The discovery of these phenomena was one of the greatest achievements of the last half of the 19th century.

Some cases of aphasia had been described before the mid-1800's, but it was Paul Broca who in 1861 began the study of the relationship of aphasia to the brain, with two major contributions.[6] He was the first to prove that aphasia was linked to specific lesions, and to show that these lesions were predominantly in the left half of the brain. The man who was, however, most responsible for initiating the modern study of this field was Carl Wernicke (Figure 1), who in 1874, at the age of 26, published his classic work, *The Symptom Complex of Aphasia*, which carried the appropriate subtitle, 'A Psychological Study on an Anatomical Basis'.[7] Wernicke established clearly the fact that there were linguistic differences between the aphasias produced by damage in the left temporal lobe, in what is now called Wernicke's area, and those produced by lesions in the frontal lobe in Broca's area (Figure 2).[8]

III. LINGUISTIC CHANGES IN APHASIA

The aphasic of the Broca's type characteristically produces little speech, which is emitted slowly, with great effort, and with poor articulation. It is not, however, only at the phonemic level that the speech of these patients is abnormal, since the patient clearly fails to produce correct English sentences. Characteristically the small grammatical words and endings are omitted. This failure persists despite urging by the examiner, and even when the patient attempts to repeat the correct sentence as produced by the examiner. These patients may show a surprising capacity to find single words. Thus, asked about the weather, the patient might

Fig. 1. Carl Wernicke (1848–1904), who, at the age of 26, published the monograph *Der aphasische Symptomencomplex*, which was to be the major influence on the anatomical study of aphasia in the period preceding World War I. During his tenure as professor at Breslau, his assistants and students included many of the later leaders of German neurology, such as Otfrid Foerster, Hugo Liepmann, Karl Bonhoeffer, and Kurt Goldstein.

say, "Overcast." Urged to produce a sentence he may say, "Weather... overcast." These patients invariably show a comparable disorder in their written output, but they may comprehend spoken and written language normally. In striking contrast to these performances, the patient may retain his musical capacities. It is a common but most dramatic finding to observe a patient who produces single substantive words with great effort and poor articulation and yet sings a melody correctly and even elegantly. Because Broca's area lies so close to the motor cortex (Figure 2), this latter region is often damaged simultaneously, so that these patients frequently suffer from paralysis of the right side of the body.

The Wernicke's aphasic contrasts sharply with the Broca's type. The patient usually has no paralysis of the opposite side, a fact which reflects the difference in the anatomical localization of his lesion. The speech output can be rapid and effortless, and in many cases the rate of production of words exceeds the normal. The output has the rhythm and melody of normal speech, but it is remarkably empty and conveys little or no information. The patient uses many filler words, and the speech is filled with circumlocutions. There may be many errors in word usage, which are called paraphasias. These may take the form of the well-articulated replacement of single sounds (so-called literal or phonemic paraphasias), such as 'spoot' for 'spoon', or the replacement of one word for another (verbal paraphasias), such as 'fork' for 'spoon'. A typical production might be, "I was over in the other one, and then after they had been in the department, I was in this one." The grammatical skeleton appears to be preserved, but there is a remarkable lack of words with specific denotation.

The Wernicke's aphasic may, in writing, produce well-formed letters, but the output exhibits the same linguistic defects which are observed in the patient's speech. He shows a profound failure to understand both spoken and written language, although he suffers from no elementary impairment of hearing or sight.

The localization of these forms of aphasia has been confirmed repeatedly. It is important to stress this point, since there is a common misconception that the classical localizations were rejected because powerful arguments were raised against their validity. The two authors whose names are most frequently quoted as critics are Kurt Goldstein and Henry Head. As I have pointed out in greater detail elsewhere,[9] Goldstein,

who had been a student under Wernicke at the University of Breslau, despite the holistic views which he expressed in his philosophical discussions, actually explicitly stated his support of the classical localizations throughout his career. Head did indeed violently attack these views early in the first volume of his famous work on aphasia.[10] His argument was, however, vitiated by the fact that, later in the same volume, the localizations which he himself supported turned out to be essentially identical to the ones he had previously dismissed as invalid.

IV. WERNICKE'S THEORY

Wernicke's contribution lay not only in establishing the syndrome patterns and their localizations but also in providing a theoretical analysis of the mechanisms of aphasia (Figure 2). He pointed out that Broca's area was located just in front of the cortical region in which lay the motor representation for the face, tongue, lips, palate, and vocal cords – that is, the organs of speech. It seemed reasonable to assume that Broca's area contained the rules by which heard language could be coded into artic-

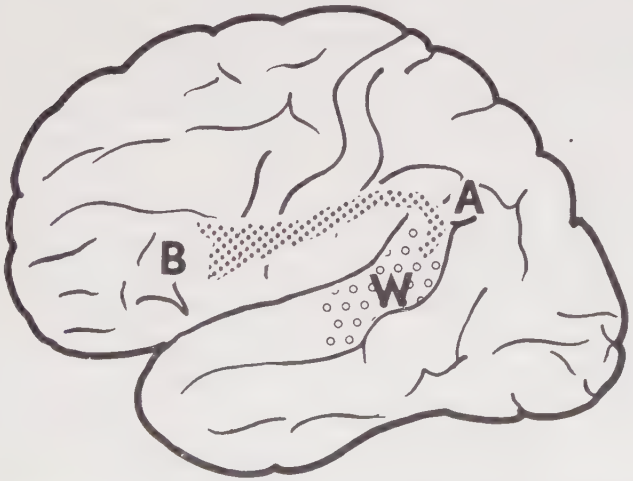

Fig. 2. Lateral surface of the left hemisphere of the human brain. *B*, Brocas' area, which lies anterior to the lower end of the motor cortex; *W* (open circles), Wernicke's area; *A* (closed circles), arcuate fasciculus, which connects Wernicke's to Broca's area. (See text.)

ulatory form. This formulation still appears reasonable. There is no need to assume that this coding need be a simple one. By contrast, Wernicke's area lies next to the cortical representation of hearing, and it was reasonable to assume that this area was somehow involved in the recognition of the patterns of spoken language. There is also no need to assume that this coding is a simple one.

Wernicke then added the natural assumption that these two areas must be connected. The general pattern was now clear. Destruction of Wernicke's area would lead to failure to comprehend spoken language. Wernicke pointed out that, for most people, written language was learned by reference to the spoken form and that therefore a lesion of this region would abolish comprehension of printed and written language. The act of speaking would consist in arousing in some way the auditory form of words, which would then be relayed forward to Broca's area to be transduced into the complex programming of the speech organs, and therefore, with damage to Wernicke's area, language output would also be disordered.

The model could readily be complicated further. Wernicke himself and those who followed him filled in further details. The comprehension of written language would require connections from the visual to the speech regions, and destruction of these connections should be able to cause isolated difficulties in reading comprehension. Since the language abilities were localized in the left hemisphere, language performances by the right hemisphere would depend on information transmission over the corpus callosum.

Clearly the validation of a theory is not a function of its surface plausibility but is dependent on other factors. It is important to remember that Wernicke's theory has been the only one in the history of aphasia which could in a real sense be put to experimental test. It was possible, on the basis of the theory, to predict that certain lesions should produce syndromes not previously described. Furthermore, it was possible, on being confronted with previously undescribed syndromes, to predict the site of the anatomical lesion. The most dramatic examples of this appear in the writings of Hugo Liepmann[11] on the syndromes of the corpus callosum. On the basis of his clinical examination he predicted the presence of callosal lesions, which were later confirmed at postmortem examination.

Several remarkable disorders of language have been described which fit readily into the Wernicke theory. In pure word deafness, the patient, with intact hearing as measured by ordinary nonverbal tests, fails to comprehend spoken language although he has essentially normal ability to express himself verbally and in writing and to comprehend written language. In this syndrome the area of damage generally lies deep in the left temporal lobe, sparing Wernicke's area but destroying both the direct auditory pathway to the left hemisphere and the callosal connections from the opposite auditory region. Although elementary hearing is intact because the right auditory region is spared, there is no means for auditory stimulation to reach Wernicke's area, and therefore the patient does not understand spoken language, although his ability to express himself in spoken and written language and his comprehension of the written language are essentially intact.[12]

In conduction aphasia, there is fluent paraphasic speech, and writing, while comprehension of spoken and written language remains intact. Despite the good comprehension of spoken language there is a gross defect in repetition. The lesion for this disorder typically lies in the lower parietal lobe (Figure 2), and is so placed as to disconnect Wernicke's area from Broca's area. Because Broca's area is preserved, speech is fluent, but abnormal. The preservation of Wernicke's area insures normal comprehension, but the gross defect in repetition is the result of disruption of the connection between this region and Broca's area. The disorder in repetition exhibits some remarkable linguistic features which are not yet explained. The disorder is greatest for the small grammatical words such as *the*, *if*, and *is*; thus, a patient who may successfully repeat 'big dog' or even 'presidential succession' may fail totally on 'He is here'. The most difficult phrase for these patients to repeat is 'No ifs, ands, or buts'. In many of these patients the ability to repeat numbers may be preserved best of all, so that, given a phrase such as 'seventy-five percent', the patient may repeat the 'seventy-five' rapidly and effortlessly but may fail on 'percent'.

V. PURE ALEXIA WITHOUT AGRAPHIA

Many examples of pure alexia without agraphia were described in the 1880's, but the first postmortem study of this syndrome was described in 1892, by Dejerine.[13] His patient suddenly developed a right visual field

defect and lost the ability to read. He could, however, copy the words that he could not understand. He was able, moreover, to write spontaneously, although the could not read later the sentences he had written. All other aspects of his use and comprehension of language were normal. At postmortem Dejerine found that the left visual cortex had been destroyed. In addition, the posterior portion of the corpus callosum was destroyed, the part of this structure which connects the visual regions of the two hemispheres (Figure 3). Dejerine advanced a simple explanation. Because of the destruction of the left visual cortex, written language could reach only the right hemisphere. In order to be dealt with as language it had to be transmitted to the speech regions in the left hemisphere, but the portion of the corpus callosum necessary for this was destroyed. Thus, written language, although seen clearly, was without meaning. This was the first demonstration of the effects of a lesion of the corpus callosum in preventing transfer of information between the hemispheres.

Dejerine's thesis has received striking confirmation. In 1925 Foix and Hillemand[14] showed that destruction of the left visual cortex in the absence of a callosal lesion does not produce this syndrome. In 1937 Trescher and Ford[15] described the first case in which a surgical lesion of the corpus callosum was shown to have a definite effect. Their patient had sustained section of the posterior end of the corpus callosum for removal of a tumor from the third ventricle. The patient could not read in the left visual field, but could read normally on the right side. This result is implied by the Dejerine theory and was confirmed by Maspes in 1948[16] and more recently by Gazzaniga *et al.*[17]. Many authors have confirmed Dejerine's anatomical findings. Michael Fusillo and I studied a patient with alexia without agraphia who demonstrated another intriguing disorder.[18,19] For approximately 3 months after his stroke he suffered from a disorder of verbal memory, which then cleared, leaving him with the reading difficulty, which remained unchanged until his death several months later. At postmortem, in addition to the anatomical findings of destruction of the left visual cortex and of the posterior end of the corpus callosum, the brain showed destruction of the left hippocampal region. It is now generally accepted that bilateral destruction of the hippocampal region leads to a permanent memory disorder. The transient memory disorder in our patient appeared to be the result of the destruction of the left hippocampal region – that is, the one located in

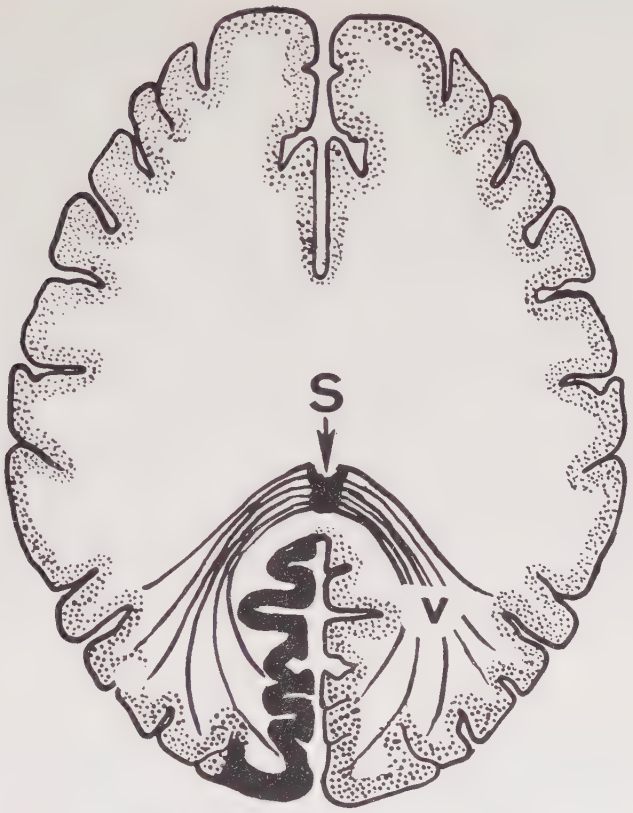

Fig. 3. Horizontal section of the human brain, illustrating the mechanism of pure alexia without agraphia; *V*, visual region. The visual cortex on the left is destroyed (heavy black line). As a result, the patient can perceive written material only in the intact right visual region. For this material to be appreciated as language it must be relayed to the speech areas on the left side through the splenium, which is the posterior portion of the corpus callosum. As a result of damage to the splenium (*S*), this transfer cannot take place, and therefore the patient cannot comprehend the written words whose form he perceives clearly.

the same hemisphere as the speech areas. Presumably it is the left hippocampal region which is necessary for the memory functions of speech cortex. After a period, the brain manages to compensate, presumably by making use of the opposite hippocampal region. Since publication of our paper,[18] I have seen several other cases of this syndrome in which memory disorder was present at the onset. It is well known that the posterior cerebral artery supplies not only the visual cortex and the posterior end of the corpus callosum but also the hippocampal region. In a certain number of cases of occlusion of the left posterior cerebral artery, all of these structures are damaged. In other cases, however, the hippocampal region is spared. Meyer and Yates[20] and Milner[4] have demonstrated that, after removal of the left anterior temporal region for epilepsy, a verbal memory disorder is observed, which is, however, generally much milder than that found in the case Fusillo and I reported, and which is not present after right anterior temporal ablation. The mildness of the disorder after left temporal ablation is probably the result of the fact that these patients had suffered from left temporal epilepsy for years and had therefore already begun to use the right hippocampal region to a considerable degree.

VI. ISOLATION OF THE SPEECH AREA

Another syndrome called 'isolation of the speech area', is explained readily by the Wernicke theory. This syndrome was described first by Kurt Goldstein[21] and has been described more recently by Geschwind *et al.*[22] We studied our patient for nearly 9 years after an episode of carbon monoxide poisoning. During this period she showed no evidence of language comprehension in the ordinary sense, and never uttered a sentence of propositional speech. She was totally helpless and required complete nursing care. In striking contrast to this state were her language performances in certain special areas. She would repeat perfectly, with normal articulation, sentences said to her by the examiner. She would, however, go beyond mere repetition, since she would complete phrases spoken by the examiner. For example, if he said, "Roses are red," she would say, "violets are blue, sugar is sweet, and so are you." Even more surprising, it was found that she was still capable of verbal learning. Songs which did not exist before her illness were played to her several

times. Eventually, when the record player was started she would begin to sing. If the record player was then turned off she would continue singing the words and music correctly to the end, despite the lack of a model. Postmortem examination by Segarra showed a remarkable lesion, which was essentially symmetrical. The classical speech area, including Wernicke's area, Broca's area, and the connections between them, was intact, as were the auditory inflow pathways and the motor outflow pathways for the speech organs. In the regions surrounding the speech area either the cortex or the underlying white matter was destroyed. The speech area was indeed isolated. The patient's failure to comprehend presumably resulted from the fact that the language inputs could arouse no associations elsewhere in the brain, and since information from other portions of the brain could not reach the speech areas, there was no propositional speech. On the other hand the intactness of the speech region and its internal connections insured correct repetition. The preservation of verbal learning is particularly interesting. In addition to the speech area, the hippocampal region, which is involved in learning, was also preserved, and this probably accounts for her remarkable ability to carry on the memorizing of verbal material.

VII. CALLOSAL SYNDROMES

Although pure alexia without agraphia[13] was the first syndrome in which damage to the corpus callosum was shown to play a role by interrupting transfer of information between the hemispheres, it was a group of Wernicke's students, including Hugo Liepmann, Kurt Goldstein, and Karl Bonhoeffer, who elucidated the full syndrome of callosal disconnection in cases in which eventually there was careful postmortem confirmation of the predicted sites of the lesions.[11,12] While the callosal syndromes continued to be recognized by German authors,[23] their existence was either forgotten or indeed totally denied in the English-language literature. In December 1961, Edith Kaplan and I presented a patient to the Boston Society of Psychiatry and Neurology who was, we believed, suffering from a callosal disconnection syndrome – a diagnosis which was later confirmed at post-mortem examination by Segarra. (Since that time several cases of confirmed callosal disconnection have been described).[24] I will mention here briefly only a few of the aspects of

our patient's condition which fit into the Wernicke theory. When writing with the right hand the patient produced linguistically correct words and sentences and carried out calculations correctly. When writing with the left hand he produced incorrect words (for example, 'run' for 'go') and performed calculations incorrectly. The theory outlined above implies that, for writing to be carried out correctly with the left hand, the information must be transmitted from the speech areas across the corpus callosum, whose interruption in our patient explained his failures. Similarly, the patient could correctly name objects (concealed from vision) which he palpated with the right hand. On the other hand he would misname objects palpated with the left hand, although it could be shown by nonverbal means that his right hemisphere recognized the object. Thus, if a pencil was placed in his left hand the patient could draw the object previously held in that hand. Again, the Wernicke theory implies that, for an individual to correctly name an object held in the left hand, the information must be transmitted from the sensory regions in the right hemisphere to the speech regions via the corpus callosum, which had been destroyed in this patient. On the other hand, the patient could read in the left as well as the right visual field. This led us to conclude that the destruction of the corpus callosum had spared the posterior end, a prediction also confirmed at postmortem.

VIII. CEREBRAL DOMINANCE

Let me turn to another bit of knowledge which fits very well into the scheme presented above. One of the most remarkable features of man is cerebral dominance – that is, the fact that in the adult the capacities for speech are overwhelmingly controlled by the left hemisphere. Out of 100 adult aphasics, at least 96 percent have damage to the left side of the brain.[25]. We do not know of any example in any other mammal of a class of learning which is predominantly controlled by one half of the brain.[26] What underlies human speech dominance? It is widely stated in the literature that the human brain is symmetrical, and this had led either to the assumption that speech dominance must reflect some subtle physiological difference between the hemispheres, or indeed even to the assumption that speech dominance is somehow acquired as the result of postnatal experience. My colleague Walter Levitsky and I[27]

decided to reinvestigate this problem, particularly since we found that some earlier authors had claimed that there were in fact anatomical differences between the hemispheres. We demonstrated that such differences exist and are indeed readily visible to the naked eye. The area that lies behind the primary auditory cortex in the upper surface of the upper surface of the temporal lobe is larger on the left side in 65 percent of brains, and larger on the right in only 11 percent. This region on the left side is, on the average, nearly a centimeter longer than its fellow on the opposite side – that is, larger by one-third than the corresponding area on the right. More recently Wada[28] has confirmed our results. He has, in addition, studied this region in the brains of infants and has found that these differences are present at birth. This region which is larger in the left hemisphere is, in fact, a portion of Wernicke's area, whose major importance for speech was first shown nearly 100 years ago. It is reasonable to assume that there are other anatomical asymmetries in the hemispheres of the human brain, reflecting other aspects of dominance.[29]

NOTES

* This article is based on a paper presented 28 December 1969 at the Boston meeting of the AAAS.

[1] I have argued elsewhere that language is based on the striking development of the angular gyrus region in man, a region which receives inputs from all cortical sensory areas (see N. Geschwind, in *Monograph Series on Languages and Linguistics*, No. 17, Georgetown Univ. Press, Washington, D. C., 1964, pp. 155–169; *Brain* **88** (1965) 237; *ibid.*, p. 585). D. Pandya and H. Kuypers (*Brain Res.* **13** (1969) 13) have shown that a forerunner of this region exists in the macaque. R. A. Gardner and B. T. Gardner (*Science* **165** (1969)) 664 and D. Premack (in a paper presented at the Symposium on Cognitive Processes of Nonhuman Primates, Pittsburgh, March 1970) have described what appears to be a definite degree of .inguistic behavior in chimpanzees.

[2] A. R. Luria, *Traumatic Aphasia*, Mouton, The Hague, 1969.

[3] O. Foerster, in *Handbuch der Neurologie* (ed. by O. Bumke and O. Foerster), Vol. 6, Springer, Berlin, 1936, pp. 1–448; W. Penfield and L. Roberts, *Speech and Brain-Mechanisms* Princeton Univ. Press, Princeton, N. J., 1959.

[4] See for example, B. Milner, in *Interhemispheric Relations and Cerebral Dominance*, (ed. by V. B. Mountcastle), Johns Jopkins Press, Baltimore, 1962, pp. 177–195.

[5] J. Wada and T. Rasmussen, *J. Neurosurg.* **17** (1960) 266; C. Branch, B. Milner and T. Rasmussen, *ibid.* **21** (1964) 399.

[6] A. L. Benton (Cortex **1** (1964) 314) summarizes the earlier literature; R. J. Joynt (*ibid.*, p. 206) gives an account of Broca's contributions.

[7] C. Wernicke, *Der aphasische Symptomencomplex*, Franck and Weigert, Breslau, 1874. An English translation has recently appeared in *Boston Studies in the Philosophy of Science* (ed. by R. S. Cohen and M. W. Wartofsky) Vol. 4, Reidel, Dordrecht,

1969, pp. 34–97. For a more complete evaluation of Wernicke's work, see N. Geschwind, *ibid.*, pp. 1–33.

[8] R. Jakobson (in *Brain Function* (ed. by E. C. Carterette) Vol. 3, Univ. of California Press, Berkeley, 1966, pp. 67–92) has given a vivid description of these linguistic differences.

[9] N. Geschwind, *Cortex* **1** (1964) 214.

[10] H. Head, *Aphasia and Kindred Disorders of Speech*, Cambridge Univ. Press, London, 1926.

[11] H. Liepmann, *Drei Aufsätze aus dem Apraxiegebiet*, Karger, Berlin, 1908.

[12] N. Geschwind, *Brain* **88** (1965) 237; *ibid.*, p. 585. There is another, less readily understood, lesion in some cases of pure word deafness which is discussed in these two communications.

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[24] N. Geschwind and E. Kaplan, *Neurology* **12** (1962) 675; M. S. Gazzaniga, J. E. Bogen and R. W. Sperry, *Proc. Nat. Acad. Sci. U.S.* **48** (1962) 1765.

[25] For a review, see O. Zangwill, *Cerebral Dominance and Its Relation to Psychological Function*, Thomas, Springfield, Ill., 1960.

[26] In *submammalian* forms there are examples of behaviors whose neural control appears to be predominantly unilateral – for example, bird song (see F. Nottebohm, *Science* **167** (1970) 950). These may represent, not an earlier stage of dominance, but rather a separate development.

[27] N. Geschwind and W. Levitsky, *Science* **161** (1968) 186.

[28] J. Wada, paper presented at the 9th International Congress of Neurology, New York, 1969.

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CHAPTER XXII

DISORDERS OF HIGHER CORTICAL FUNCTION IN CHILDREN

PREFACE

The great majority of these who concern themselves with disorders of the higher functions in children are usually interested in the developmental disorders of language, reading, and other behaviors. I have myself not had a special interest in this group of disorders, because of my lack of extensive clinical experience with them and my generally limited contact with pediatric neurology, but probably even more because of the lack of strong anatomic data about these conditions. I have the suspicion that the neurological substrate of many of these cases will be neither damage to structures, nor gross congenital malformations,* but rather the small size of certain brain regions. This speculation, however, remains to be confirmed or rejected.

The paper presented here therefore deals almost exclusively with the acquired disorders of children, for which the comparison with adult lesions is more fruitful.

It should be remembered that when one speaks of better recovery from childhood lesions two somewhat different circumstances are under consideration. On the one hand the early lesion, in, let us say, the speech area itself, is followed by excellent recovery. On the other hand it must also be remembered that the patient with an early lesion will recover better from certain later lesions. Thus many of the cases who have undergone cortical excisions for epilepsy will show relatively mild syndromes, since these patients nearly all have had early lesions. Similarly nearly all the cases who have undergone surgical section of the callosum have been patients with epilepsy dating back to childhood and have had milder syndromes than those seen in adults with vascular disease. In this case it appears that the presence of an early lesion almost anywhere in the brain probably makes for a milder syndrome in response to a later callosal section.

DISORDERS OF HIGHER CORTICAL FUNCTION IN CHILDREN*

1972

I am very honored to be invited to speak to you as the Richmond Paine lecturer. My own personal contact with Dr. Paine began over 20 years ago when I was in my final year in medical school and taking my pediatric rotation at the Children's Hospital in Boston where Dr. Paine was then a resident working on Dr. Bronson Crothers' pediatric neurology service. To this day I remember two superb and scholarly conferences that Dr. Paine gave which were my first introduction to pediatric neurology, and which I still quote. One was a talk on facial paralysis in childhood and the other one, which was particularly intriguing, was on heterochromia iridis. People find it still interesting when I quote Dr. Paine, to hear not only about humans with discordant pigmentation of the irises, but also about cats with one blue eye, one green eye, and unilateral albinism. It is also a particular pleasure for me to be invited here by Dr. Michael Malone who carried out his neurological training in my unit.

It is with mixed feelings that I come here since I am not a pediatric neurologist nor, in fact, have I had an extensive experience with children with disorders of the higher cortical functions. Despite this, however, I would like to suggest to you that that those who are concerned with the developmental or acquired disorders of childhood might find it very useful to know what has been learned about adults with disorders of the higher cortical functions. Similarly, I hope that I will convince you that those of us who are primarily concerned with adult disorders cannot afford to neglect the insights which can be obtained from the study of childhood disturbances. It would seem obvious that what is known about the adult should be important for the understanding of the child and vice versa. The fact is that there is still very often a tendency for those working in the adult and childhood disciplines to isolate themselves from each other. I hope to show you some ways in which these fields can stimulate each other.

My own approach to the problem of the higher functions has tended to be one which had for long been regarded as completely outmoded. I

have been primarily concerned with the relationship of the higher functions to the structure and the physiology of the nervous system. This is by no means the only valid approach but I feel it would be impossible to advance the field without this approach being taken into account in a major way. I do not know of any branch of medicine in which looking inside the 'black box' has not in the end paid off, and I think this is an indispensable approach to the higher functions.

I would like to highlight some of the differences between the acquired aphasias of adults and those of childhood. To do this I will first point out some major features of the acquired aphasias of adults (Geschwind, 1970). Most of us were taught a certain classification of aphasia which is perhaps still the most widely taught classification in the United States. It is unfortunately not useful, and furthermore, although many people have the mistaken notion that this is a classical classification, it is in fact a very recent invention which is not based on any adequate evidence in the literature. This view states that aphasic patients are divided into two categories. The first includes those patients who have trouble speaking, i.e., an expressive difficulty which is called expressive aphasia. According to this view, the lesion in expressive aphasia is in Broca's area. On the other hand, a patient who has a pure comprehension difficulty with language is said to have a receptive aphasia; if the patient has both, he has a mixed aphasia. According to this view, most patients have mixed aphasias. The lesion for pure difficulty in comprehension of spoken language is said to lie in Wernicke's area. In fact Wernicke did not say this, although to this day, this erroneous view is attributed to him again and again.

This classification of 'expressive aphasia' meaning difficulty in talking (and thus a lesion in Broca's area) and 'receptive aphasia' meaning difficulty in understanding language and thus a lesion in Wernicke's area, does not fit the classical notions of the clinical pictures and has no support from what has been learned from postmortem studies of aphasia. There is a very common notion that there was a great disagreement about the localizations of aphasic syndromes. In fact it is commonly accepted that these disagreements continued until Henry Head (1926) came along and showed that there was chaos in the field. It turns out, however, that Head's own localizations, in the very same book, are in clear agreement with the classical authors whom he criticized so violently.

Head's book is in fact self-contradictory. I cannot in fact find any instance in the history of aphasia where anyone has studied the correlation between the clinical pictures and localization and not come up with essentially the same answers. Wherever the contrary assertion has been made it has been advanced without the support of the neuropathological evidence in properly examined patients.

Figure 1 is a diagram showing Wernicke's and Broca's areas. Patients with both Broca's and Wernicke's aphasias have trouble speaking. The Broca's aphasic, however, tends to produce very little speech, which is poorly articulated, uttered with great effort and with abnormal rhythm and melody. His disabilities, however, are not confined merely to the effortful production of disarticulate speech because the Broca's aphasic also shows certain features which can only be described as disorders at the level of language, or linguistic abnormalities. The Broca's aphasic does relatively well in naming things and producing single substantive words but does poorly in putting the grammatical skeleton on the sentence, not only in spontaneous speech but also when repeating. He produces grammatical words more poorly than nongrammatical ones and his speech tends to take on the quality of a telegram. The Wernicke's aphasic also has trouble speaking but his speech abnormality is of a totally

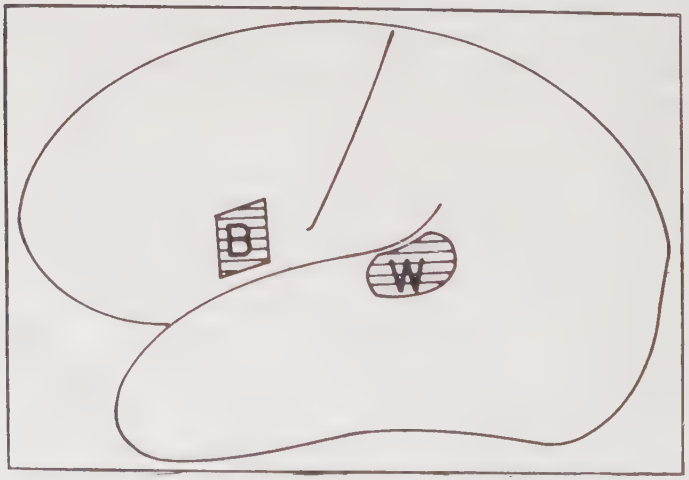

Fig. 1. Wernicke's and Broca's areas.

different variety. He produces speech which if it is not listened to carefully or it it is in an unfamiliar language, one would think was normal speech. It has normal rhythm, melody, intonation, and the sounds are correctly articulated. The patient speaks rapidly and, in fact, in the extreme cases, he speaks more rapidly than normal. As he improves, his speech slows down. Furthermore, when listening to the speech of the Wernicke's aphasic, you will find that it has a normal grammatical skeleton and contains the small grammatical words in the proper context. The patient's major difficulty is in finding the correct word as he speaks. He may sometimes leave a word out or produce pauses which give a spurious impression of nonfluency, or he will use incorrect words with correctly articulated sounds.

Which of these forms of difficulty in speech output can be called expressive aphasia when they are so different? If you accept the simple argument that 'expressive' difficulty means a lesion in Broca's area, you will often be wrong. The word expressive aphasia classically meant only Broca's aphasia, not the expressive disorder of Wernicke's. The word 'expressive' should be abandoned because it only leads to confusion. It is preferable to speak about fluent aphasias such as those seen with posterior lesions, the Wernicke's aphasia being the characteristic variety of this type; and nonfluent aphasias, such as are produced by lesions in Broca's area.

One of the first and most striking differences between the child and the adult is that while fluent aphasias in the adult are very common and make up a sizable proportion of all aphasias, there is in the child a striking lack of obviously fluent aphasias. I have been told but have never personally verified the statement that this observation was first made by Freud. The curious thing is that even with lesions which in the adult would typically lead to fluent aphasias, the child does not suffer from an obviously fluent aphasia. This introduces a problem clinically in that the differential diagnosis of the aphasias on the basis of the quality of the patient's output is much more difficult in childhood than in adult life.

While my own experience has been with small numbers of acquired aphasias in childhood, I think that those who have had considerable experience with adult aphasics can detect in occasional cases in childhood the essential but much less marked characteristics of fluent aphasia, but never the gross logorrhea that is frequently seen in the adult. This is obviously a major difference between the adult and the child and

it is not fully clear why it exists, although we can speculate. The presence of a fluent aphasia guarantees the intactness of Broca's area because a Broca's lesion can only produce a nonfluent aphasia. It is thus possible that a fluent aphasia represents the free running of an intact Broca's area. Perhaps the child's Broca's area has not had adequate practice in language to run on as freely as that of the adult. There is some support in this view from several sources. Studies made in adults (Isserlin, 1936) have shown that the appropriate lesion at different stages in adult life will tend to produce more fluent aphasias the older the patient is, i.e., a patient of 50 with lesion in Wernicke's area is much more likely to have a grossly fluent aphasia than a man of 25. Another fact which fits in with this interpretation is that it is much easier to tell aphasias apart in the adult on the basis of their speech but not of their writing. Both Broca's and Wernicke's aphasics write abnormally. The reverse, by the way, is not true; you can have a patient with aphasic writing who speaks normally but I have never seen a patient who had aphasic speech and wrote normally. It is much harder in the adult to tell aphasics apart on the basis of their writing, since writing tends to be sparse in all aphasics, and truly 'fluent' agraphias do not occur. Any patient who produces pages and pages of abnormal writing is almostly certainly not aphasic, but this type of abnormality is occasionally seen in schizophrenics. This absence of aphasic graphorrhea would seem reasonable in that all humans have spoken an enormous amount by the time they get to adulthood, but even people who write a lot will relatively have produced much less written language than a normal child would have by age 10.

Another important point is that the adult aphasic is not very often mute. Many people tend to think that aphasia means lack of speech. This is an inadequate description because the adult fluent aphasic may speak more rapidly than normal. The adult with Broca's aphasia is sometimes mute in the acute phase but later on, despite a grossly constricted output, is usually not mute. The child with an acquired aphasia is, however, much more likely to be mute and this may present a problem in diagnosis. If a patient does not speak at all it may be difficult to tell if he is aphasic because one of the criteria for aphasia is that the patient is producing abnormal language. Many mute adults do not have lesions in the speech areas.

Mutism in the child presents even more of a problem. Children may

become mute under many circumstances. On the one hand, they share all of the causes of mutism on the basis of lesions of the nervous system; they are however, much more likely to become mute under situational stress. Thus a child who comes into the hospital to have an appendectomy may fail to speak after the operation. It is usually easy enough in this situation to be certain that there is no lesion in the nervous system, but what about the child who is brought in with a head injury and does not speak. Is he aphasic or not? What about the child who has had a neurosurgical procedure and does not speak afterwards? There is one method that is often effective in distinguishing mutism on the basis of situational stress from that resulting from lesions to the nervous system. Usually the child with psychological mutism will recover rapidly after having been home for a few days.

If an adult patient is mute but writes normally, you can be almost absolutely sure that he is not aphasic. By writing normally I do not mean just being able to sign his name, but rather the ability to express himself fully in writing. It is sometimes difficult, however, to get the mute child to cooperate in writing. If the child wrote normally, my own guess would be that the same rule held true – he would not be aphasic.

The most important difference between the child and the adult is the much more dramatic recovery of the child from aphasia. What is the mechanism of this? This dramatic capacity of the child for recovery often leads to the assertion that this proves that those who have tried to portray the brain as some kind of simple machine are wrong and that the nervous system is a device of marvelous plasticity. Unfortunately this assertion tells us little except what we knew already, i.e., the child recovers better than the adult. The whole notion of plasticity often has a rather unfortunate effect because it leads to failure to identify mechanisms. Indeed those who look for mechanisms are often accused of denying the plasticity of the nervous system. My argument is however, that if you were to accept plasticity as a kind of theological given, you would never understand its mechanism. The nervous system is, however, unfortunately not always plastic, as the vast numbers of permanent severely adult aphasics prove. If we understood the mechanisms at play in those cases in which plasticity is manifest we may be able to help those in whom it is not. It is thus most important for us to consider the possible means by which the child recovers so well.

The answer often given is that the child tends to recover so well from aphasia because he relearns with the right hemisphere, but I suspect that this statement is incorrect. I do not object to the notion that these children use the right hemisphere for language, but rather to the notion that they have *relearned* language with the right hemisphere. The implicit notion in the usual argument is that the child first learned speech with the left hemisphere and, after damage, relearned language with the right hemisphere. It is usually argued that the adult recovers more poorly because his right hemisphere lacks the capacity for relearning. There is, however, strong evidence against relearning with the right hemisphere because the speed with which the child recovers in many instances from even the most massive left hemisphere lesions is incompatible with a relearning process. I have, for example, seen a patient who had what Donald Matson (personal communication) described as the largest glioma he had ever seen and in whom a large left hemisphere resection was carried out. This child was left with a permanent right hemiplegia and was very severely aphasic after the operation. He was still severely aphasic a month later; by two months he had a considerable amount of speech; by three months he was speaking normally. It is inconceivable that the right hemisphere of the child is capable of relearning English to that degree of complexity within three months. Furthermore, at the time of recovery he was fully capable of discussing his past life. It would be difficult to accept that during a three month stay within the hospital he had somehow reacquired all his previous vocabulary. One must advance a different kind of explanation, i.e., that this was not relearning by the right hemisphere, but that, in fact, the right hemisphere must have been learning language all along. The child's previous left hemisphere dominance must have reflected not exclusive learning by the left hemisphere but dominance in the literal sense, i.e., it was the one which was performing both in terms of comprehension and speaking of language. After its destruction, the right hemisphere eventually was able to come to use the learning which it had been acquiring all through childhood. What is not clear is why it should take several months for this capacity to become manifest. Exactly the same thing can be seen in the occasional adult who recovers from a massive, almost total, destruction of one hemisphere. There are cases in the adult where one hemisphere has been almost entirely destroyed and the patient has been aphasic for many months, but has eventually

undergone a major recovery, eventually speaking practically normally and clearly using the other hemisphere (Dejerine, 1912). A relearning mechanism again is excluded, since it is inconceivable that someone at the age of 60 should reacquire a language to a high degree of skill within six months to a year and regain his old vocabulary. Adults do this much less frequently than children, and those who do so tend to be either left-handed or have strong family histories of left-handedness (Luria, 1970). The adult left-hander appears to be much more similar to the child in these respects. The only hope, in my mind, for really effective rehabilitation of the adult aphasic depends on the possibility that he, like the child, has language learning in the other hemisphere which for some reason he is not capable of using. It would clearly be a remarkable advance if every adult aphasic could recover to the level of speech of the 9 year old. The number of permanently severe aphasic patients who do not recover even close to that level is enormous. Here is an instance where understanding what is going on in the child is vitally important. If we knew the mechanisms involved in the child, perhaps some way could be found to 'attack' the right hemisphere of the adult in order to make the latent language learning become manifest.

There are, furthermore, some differences in the mode of recovery in the child and the adult. In the child who recovers from aphasia, although his speech may now be normal, he has difficulties when formally tested on specific grammatical areas (e.g., one horse, two –?). This is different from the adult who recovers to the same extent; you will not usually find this sort of special late grammatical difficulty.

There are some other related features of recovery in childhood that deserve comment. There has been a tendency to assume that the child who recovers from an aphasia has returned to full normalcy. Hebb (1942) argued on the basis of studies of brain lesions at different ages that the earlier the brain damage the more likely was one to observe later a generalized decline in performance in all spheres, while the later the age of injury the more likely one would observe selective effects. Byers and McLean (1962) followed 10 cases of childhood aphasia for several years. All regained normal speech spontaneously without special retraining. Of these, seven, however, showed distinct impairments on psychological testing, four of them in visuomotor and spatial functions. In other words, like Hebb, these authors found that whereas in adults, damage to speech

areas often leads to permanent aphasia, the child with the same lesions often appears to 'trade' specific language disorder for a deficit in nonverbal functions. Lansdell (1969) argued, on the basis of his own careful studies, that in patients with early left hemisphere damage which led to a shift of speech to the right hemisphere, the earlier the damage the more likely one would find in later years normal verbal processes and a deficit in nonverbal processes. He argues, reasonably, for the notion that it is the competition for right hemisphere tissue which suppresses the full development of nonverbal capacities. We can thus see that the child often pays a price for the ability to recover from aphasia. On the other hand there may be clues in these data to aid us in understanding the mechanisms of recovery.

Implicit in the discussion up to this point, and implicit in many discussions is the view that brain lesions in childhood produce less serious effects than lesions in adult life. There are clearly many instances in which we know this to be true. Thus the child who undergoes a large cerebellar removal generally recovers very well, while the adult is often left with permanent severe cerebellar signs. The same is true as we have already noted for most childhood aphasias. We must be wary, however, of assuming that this is a general principle. Thus it appears at least possible that some childhood lesions may produce aphasias while the same lesions do not do so in adults. Let us now consider one possible example of this.

The patient with adult Korsakoff's syndrome suffers from two major intellectual difficulties (Victor *et al.*, 1971). He has trouble in learning new things and he usually has a retrograde amnesia extending back several years before the onset of the illness. He may otherwise be perfect intellectually. Although the retrograde amnesia may extend back for many years, so that the patient may deny being married or having children, some kinds of memory appear to be preserved, e.g., aphasia is never seen as a part of the adult Korsakoff syndrome. One answer might simply be that the memory deficit was selective, but this would clarify little. Further examination of the memory deficit may help us more. Thus you will find that on the whole this patient tends to preserve relatively better those things which he learned before the age of 12. Even if his retrograde amnesia extends back 20 or 30 years, he usually remembers the names of his parents, where they were born, the names of his elementary school teachers, where the school was, his address as a child and a whole array

of similar facts. If what was learned before 12 is better preserved, then it certainly would be reasonable that he would not be aphasic since the major period of language learning also occurs before the age of 12.

But then, what happens when you see a Korsakoff's syndrome in childhood? Curiously, although this syndrome is fairly common in adult life, my experience has been that pediatric neurologists do not see it. It is true that thiamine deficiency Korsakoff's syndrome would be unlikely in children. Even in the adult the most common cause of Korsakoff's syndrome is not metabolic disorder but head injury, leading typically to a transient Korsakoff's syndrome. Despite the frequency of head injuries in childhood, descriptions of Korsakoff's syndrome at this age are very rare, if indeed they occur at all. One possibility is that the syndrome occurs but is not recognized because the clinical picture is different in the child. This speculative possibility was raised by an experience I had some years ago. I saw a patient who at age 10 had developed some mental symptoms and who was referred to a physician who subjected the child to an extensive course of shock therapy. Because his condition was getting worse, he was transferred to another institution where shock treatment was discontinued, and over the next few months be began to improve. When I first saw him I found that he walked normally, had full strength and coordination throughout, and that all his sensory systems were operating quite well. However, he ate with his hands, had a great deal of trouble dressing himself, and was obviously aphasic in speech. In addition to having severe comprehension deficits, he had, to the extent that he could be tested, severe memory deficits as well. As he improved further, it became very clear that he was suffering from the combination of aphasia and memory deficit. One's first tendency would be to assume that the boy had suffered 'diffuse brain damage', but this is a diagnosis I always find difficult and which was particularly hard to accept in this case. How could the nervous system be affected diffusely in a patient who showed a total lack of elementary neurological disorder? It seemed clear that this disorder must be selective.

In the adult who is given excessive electric shock, one typically gets a Korsakoff's syndrome, and pathological changes can be found in the medial temporal regions of such patients. Is it conceivable that this child was suffering from a Korsakoff's syndrome? If so, why then was he aphasic? Perhaps the reason is that in some cases of childhood Korsa-

koff's syndrome the retrograde amnesia includes the period of acquisition of language and therefore aphasia and, indeed, many other disorders are seen that do not occur in the adult. It is easy to see why, if this were the case, Korsakoff's syndrome might not be recognized in childhood and such cases might be labelled as examples of diffuse brain damage.

The speculations raised here have other implications. Consider for example the group of children who are mentally defective but have a normal brain in terms of total weight. Is it possible that some of these are congenital Korsakoff's syndromes, resulting from a defect in the systems involved in laying down memory? If this occurred, then despite the intactness of the rest of the brain, the child could be seriously defective. Some well-localized biochemical lesions in this system might be all that was required to produce so devastating a clinical picture.

In adults there are transient aphasias (Ciemins, 1970) but there are no permanent cases of aphasia from lesions in the thalamus to my knowledge. Is it conceivable that thalamic lesions in the child might produce aphasia by interfering with language acquisition, although in the adult the same lesion, once language is acquired, produces only minor permanent effects?

Let me now turn to another aspect of the brain – cerebral dominance – which has always been of particular interest to those concerned with development. In recent years it has been commonly stated that dominance has no obvious anatomical basis (Bonin, 1962). We have, however, shown that cerebral dominance is not based on some ultra microscopic difference between the two hemispheres or on some subtle physiological difference, but is probably the result of a gross anatomical difference between the hemispheres readily visible to the naked eye (Geschwind and Levitsky, 1968). Levitsky and I took up this investigation in part because we felt that the rejection of an anatomical basis for dominance had been made too lightly, particularly since some very distinguished people had insisted that there were anatomical differences (Pfeifer, 1936). We took 100 brains and made a section running through the sylvian fossa so we could look at the upper surface of the temporal lobe on the two sides. The area we looked at was the region lying behind Heschl's gyrus and the back end of the sylvian fossa. This area is called the planum temporale.

In the 100 brains we examined, we found that the left side was larger in 65, the right larger in 11, with approximate equality in 24. This

difference was significant at better than the 0.001 level. Although the left side was often dramatically larger than the right, the reverse was rarely true. In terms of absolute measurements in the 100 brains, the left side was one-third larger than the right and the average length of the left side was nearly a full centimeter longer than the right, another difference statistically significant at better than the 0.001 level. There is no asymmetry like this known in any other animal of the mammalian series. The area which is larger on the left turns out in fact to be part of Wernicke's area, probably the most important of the speech regions of the hemisphere.

Since we published this study, Wada (1969) in Vancouver has repeated this study in adults, confirming our results with similar statistics. He has also carried out this study on the brains of newborn infants and fetuses and found that the differences we had described in the adult were present to the same degree at birth. Thus these differences are not the result of life experience but are present even in utero.

Let me turn away briefly from the acquired disorders of childhood to that whole array of so-called developmental disorders. I think that to treat all of these special learning deficits as abnormalities is probably incorrect in most instances, and that most of these children are normal variants of the human species. One must remember that practically all of us have a significant number of special learning disabilities. In most cases these disabilities do not get us into difficulties, but this is merely cultural accident. For example, I am grossly unmusical and cannot carry a tune. A significant number of children are unmusical despite valiant attempts to teach them, while other are obviously highly endowed despite total lack of any special instruction. Some children similarly are very artistic and can draw beautifully, and others, like myself, are quite incapable of drawing even reasonably. Such difficulties are quite widespread throughout the human race, yet we, the unmusical minority and the unartistic majority, are not labelled as suffering from 'minimal brain dysfunction'. As in most species of animals, talents are distributed asymmetrically. We happen to live in a society in which the child who has trouble learning to read is in difficulty. Yet we have all seen some dyslexic children who draw much better than controls, i.e., who have either superior visual perception or visual motor skills. My suspicion would be that in an illiterate society such a child would be in little difficulty and might in

fact do better because of his superior visual-perceptual talents, while many of us who function well here might do poorly in a society in which a quite different array of talents was needed to be successful. In most cases, we probably are not talking about disease but about children who have run up against the demands of this society for particular sets of talents. As the demands of society change, will we acquire a new group of 'minimally brain-damaged'?

How do we account for these variations ? If indeed these are not diseases, then would you not expect to find that the brains of these children were normal? Yes, they would be normal but I believe they would still be different from one another. Generally when the neuropathologist looks at brains of children he is searching either for obvious congenital anomalies or for microscopic pathology. Yet brains which differ dramatically may show no changes of either of these types. For example, there is to our knowledge no significant microscopic difference between the planum temporale on the right and left sides, but one cytoarchitectonic area is simply much larger on the left (v. Economo and Horn, 1930). Gross differences in the sizes of areas on the two sides may be exceedingly important in brains which are normal. What happens to the child whose brain shows a bilateral right-sided pattern of the planum temporale? What is that child like as compared to one who has a huge left planum and a small one on the right side? In other words, there are many different variations here which may well correspond to differences in talent. I suspect that there are innumerable other gross anatomical differences between the hemispheres that we have not yet looked at and which indeed are not easy to see.

Lemay and Culebras (1972) have recently published the impressive results of a recent study in which several further asymmetries are evident. They found, in conformity with older observations of Cunningham, that the right sylvian fissure is angled upward more than the left, but also made the new observation that the left parietal operculum is better developed than the right. The study of Levitsky and myself was perhaps most surprising in that the gross differences we found were so striking. Once we have learned to examine the normal brain grossly, we may have a powerful means for finding the source of those remarkable variations in perceptual and learning capacities in different areas which exist in normal children.

NOTES

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CHAPTER XXIII

WRITING DISTURBANCES IN ACUTE CONFUSIONAL STATES

PREFACE

Disorders of writing present a paradoxical problem to the student of the higher functions. Thus the evidence known to me at this time leads me to believe either that there is no localized cortical lesion which can produce a bilateral disorder of writing without aphasia in spoken speech, or that such lesions must be extremely rare. On the other hand cases in which disorder of writing is the unique or predominant linguistic disorder are not at all rare. I had for some years been struck by the high frequency of writing disorders in confusional states, and this paper with Dr. Chédru confirms this finding with both spontaneous clinical cases and experimental cases. We have no good idea as to why disorder of writing should be so prominent in metabolic or toxic disorders. There are other disorders which occur in confusional states, as Chédru and I have discussed in another paper, although they have not been studied as well as writing disorders. One might suspect that the functions disturbed are those which depend most critically on coordination of different parts of the brain and on the proper operation of the mechanisms of attention, which are perhaps the most complex of all functions of the brain.

WRITING DISTURBANCES IN ACUTE CONFUSIONAL STATES*

1972

ABSTRACT. Studies of writing ability were carried out in 34 acutely confused patients. Their performances were compared to those of 10 controls and, in 24 of the cases, to their own performance after recovery from confusion.

Writing was impaired in 33 of the 34 cases. The writing disorder could involve the motor and the spatial aspect of writing as well as spelling and syntax. It was the most constant and the most striking linguistic disorder seen in these patients. It disappeared when the confusion cleared. The spelling disorder had the following features: high error rate in consonants and of small grammatical words in their entirety, high rate of omission and substitution, high involvement of the last letters of the words.

The problem of pure agraphia is discussed in the context of these findings.

Writing disturbances in focal brain lesions are well recognized (Marcé, 1856; Benedikt, 1865). In general, such disturbances are associated with aphasic, alexic or apraxic signs (for a recent review, see Leischner, 1969). In a few cases, however, writing impairment has appeared to be isolated. This isolated defect in graphic expression has been called 'pure agraphia' (P.A.). In these cases the lesion has most often been considered to be localized in the left frontal area, (Gordinier, 1889; Henschen, 1922). For these authors, such cases represent a confirmation of the cerebral writing center postulated by Exner (1881) to be in the foot of the left second frontal convolution.

Many neurologists have however been reluctant to accept the existence of such a center, (Dejerine, 1891; Wernicke, 1903; Rawak, 1933; Russell and Espir, 1961; Hécaen *et al.*, 1963). The present study was in part motivated by our own skepticism concerning the existence of pure agraphia on the basis of an isolated destructive lesion in the left frontal lobe. We had been struck first by the difficulty of finding such cases (either personal or in the literature). Secondly we had found that, in contrast to our failure to find cases of pure agraphia with focal vascular disease, we had seen many cases of agraphia, with little or no other language disorder, in patients with toxic or metabolic confusional states. Thirdly, we had noted that the cases of P.A. in the literature, although often attributed to focal disease, frequently

had tumors and were often stated to show general mental impairment.

We therefore decided to explore methodically the question as to whether isolated writing disturbance could be found in cases suffering from acute confusional states (A.C.S.).

For the purposes of our study, we have characterized the principal features of the A.C.S. as follows. The main characteristic is a reduction and/or a ready shifting of attention. Other symptoms, (e.g. disorientation, changes in mood, hallucinations), are not essential for the diagnosis. A.C.S. may occur in any kind of toxic or metabolic disorder, especially of rapid onset, as well as in cases of head injury, subarachnoid hemorrhage or increased intracranial pressure, especially when of rapid onset. The type of pathogenic agent involved appears to have little bearing on the clinical picture of the A.C.S. (Bonhoeffer, 1912).

In this report we will describe the writing impairments we observed in 34 acutely confused patients. On the basis of these observations, we will reconsider the problem of P.A.

I. METHODS

Subjects

Four groups of subjects were examined: (1) Patients presenting with 'natural' A.C.S., (2) patients recovering from electro-convulsive therapy, (3) Patients receiving barbiturates during the induction of general anesthesia, (4) controls. For the pathological groups, the following requirements were set:

(a) reduction and/or shifting or attention of recent onset related to a well defined toxic or metabolic disorder. The attention disorder was evaluated on the basis of clinical examination.

(b) absence of signs of focal brain lesions and of chronic intellectual deterioration.

(c) age below 60 years.

(d) ability to carry out the test battery.

Every patient – but 3 of group 2 – demonstrated some disorientation for time and place. On the other hand, every patient of groups 1 and 3 performed abnormally on one or both of two tests generally considered as 'attention': digit span and a test of auditory – motor attention (patients of group 2 were not submitted to these tasks).

Group 1: Patients presenting with 'natural' A.C.S. The etiologies were: delirium tremens (19 cases), hepatic encephalopathy (6), Wernicke's encephalopathy (4), acute alcoholic intoxication (1), barbiturate intoxication (1). In several cases more than one disorder was present.

Group 2: Patients undergoing electro-convulsive therapy (E.C.T.). These patients were examined during the short confusional state following E.C.T. Before E.C.T., each patient received barbiturates and the subsequent A.C.S. may have been related both to medication and E.C.T.: 9 patients were suffering from psychotic depression and 1 from schizophrenia.

Group 3: Patients undergoing general anesthesia. Two patients were studied while under the effect of I.V. barbiturates (total doses: 400 and 500 mg of Amytal), administered as normal premedication before surgical anesthesia. They remained under the effect of Amytal throughout the interview.

Group 4: Controls. The *S*s were chosen from the medical wards. They met requirements (b), (c) and (d) above and were not confused. No attempt was made to exclude alcoholics.

The characteristics of the 4 groups in regard to number of *S*s, age and educational background are given in Table I.

TABLE I

Group characteristics (number of *S*s, age, educational background). Educational levels were defined as follows: Level 1: *S*s having had no high school eduction. Level 2: *S*s with more than 1 year in high school, but not graduates. Level 3: high school graduates.

	Number of *S*s	Age (mean age and range)	Educational background levels		
			1	2	3
Group 1 ('natural' A.C.S.)	22	44 (28–55)	11	3	8
Group 2 (E.C.T.)	10	46 (24–60)	3	3	4
Group 3 (general anesthesia)	2	42 (37–44)	1		1
Group 4 (Controls)	10	41 (24–59)	3	3	4

Procedure

The writing tasks consisted of the following:

(a) writing *to command*: patients were given the command to compose and write two sentences, one about the weather, another about their jobs.

(b) writing *to dictation*: six words (business, president, finishing, experience, physician, fight) and two sentences ('the boy is stealing cookies', 'if he is not careful the stool will fall') were presented. Several presentations were often necessary.

(c) writing *to copy*: the patient had to copy in script a sentence written in printed letters ('the quick brown fox jumps over the lazy dog').

Self-correction was discouraged but could not always be prevented. Occasionally the *S*s were given block letters and asked to compose 2 of the words that had been dictated. No time limit was set.

This study was carried out within the framework of a larger study on disorders of higher cortical functions in A.C.S. (Chédru and Geschwind, 1972). Other language tests were thus given including spontaneous speech, naming, comprehension, spelling, repetition, and a wordlist test. In addition, evaluations were made of behavior, affect, attention, the other components of the Gerstmann syndrome and praxis.

Subjects of groups 2 and 3, as well as 12 subjects of group 1 were tested twice, once during the A.C.S. (examination A), and a second time when the confusion cleared (examination B).

II. DATA

Writing was frequently impaired in regard to its motor, spatial and linguistic aspects. The high rate of writing impairment is shown by the fact that, of the 34 confused patients, only one did not demonstrate any abnormality in writing while in A.C.S.

Motor Impairment

Of the 34 subjects with A.C.S., there were 10 who wrote the letters normally but with a mild tremor (e.g. cases 4 and 20 – see Figures 1 and 2). In 5 cases, writing was reduced to an illegible scribble (e.g. cases 6 and 8 – see Figure 1). In the remaining 19 cases, writing performance fell between these two extremes and manifested some or all of the following

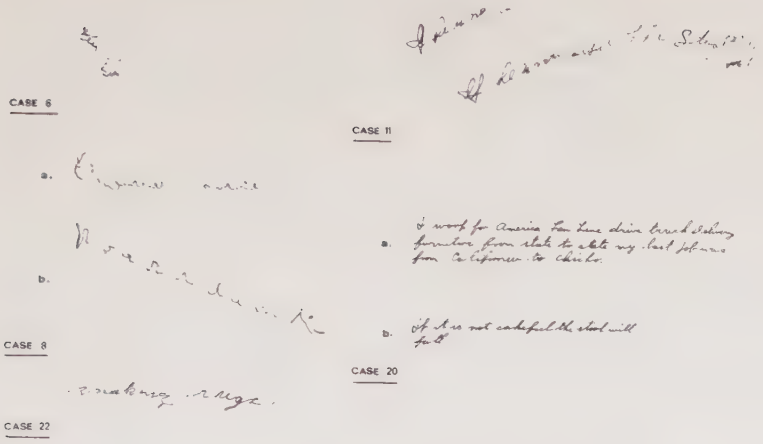

Fig. 1. Samples of writing performances of actuely confused subjects.

Case 6: 'finishing' (dictation). *Case 8:* 'president' (dictation). (a) first attempt; (b) second attempt; the patient had been told at this time that he could write in print if he wished. *Case 22:* description of the patient's job (to command). *Case 11:* 'if he is not careful, the stool will fall' (dictation). Two attempts were necessary because the subject was writing too close to the margin of the sheet. *Case 20:* (a) – description of the job (to command); (b) – 'if he is not careful...' (dictation).

Writing to command means the composition by the patient of a sentence on a specific subject set by the examiner.

The samples of this picture have been redrawn with India Ink for purposes of reproduction.

disorders: letters clumsily drawn; absence of loops of the *l*, *f*, *g*, and *o*; reduplication of strokes in letter such as *m*, *n*, *w*, and *u*; no dots on the *i*'s; letters overlapped or not linked. Sometimes contamination from a neighboring letter produced curious 'neographisms' (e.g. case 22 – see Figure 1). To be noted is the absence even in the severest impairments seen by us, of reiterated drawing of curves, loops and circles, such as in frequently reported in cases of P.A. (Gordinier, 1899; Campbell, 1911).

Printed letters appeared to be easier to write. In some cases, the *S*s improved when switching from script to print. In 6 cases, the *S*s either could not switch to script when asked or mixed script and printed letters (e.g.: case 4 – see Figure 2). Micrographia was present in 3 cases. In most of the performances no punctuation marks were used.

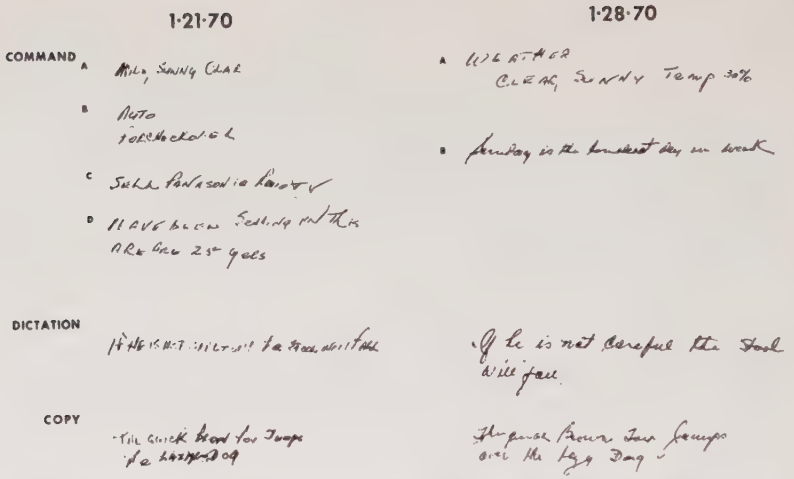

Fig. 2. Samples of the writing performances of a patient (case 4) during (left of illustration – 21 January 1970) and out of (right of illustration – 28 January 1970) an acute confusional state.

Spatial Disorders

Twenty-three *S*s could not align letters properly and oriented the lines upward or downward. Some wrote close to the margin of the sheet, demonstrating something comparable to the 'fear of emptiness'' mentioned in focal parietal brain lesions (Critchley, 1953). In copying words, 4 patients wrote in close proximity to the model, sometimes overlapping their letters with those of the model.

Reluctance to Write

19 of the confused *S*s consistently demonstrated reluctance to write, alleging: "I never could write well, you know...," "I am not much of a writer," "without the proper glasses, it's difficult...."

Syntactical Disorders

Fourteen *S*s, although having been asked repeatedly to write in *full* sentences, and apparently having understood what the examiner meant, wrote 'headline' type sentences such as 'weather fair', 'making rugs', 'sell T.V.'... Such agrammatic answers were in striking contrast to the

normal grammatical structure of the oral expression of the same patients.

A similar discrepancy between oral and written grammar has been described by Hécaen *et al.* (1963) in conduction aphasia.

Spelling and Other Linguistic Errors

These were found in most cases even when there was no motor or spatial difficulty (e.g. cases 4 and 20 – see figures).

Comparisons of the written spelling performances during and after A.C.S. were carried out for 21 *S*s (10 *S*s of group 1, 9 of group 2 and the 2 subjects of group 3). These comparisons are summarized in Tables II and III. During A.C.S. *S*s made about three times as many errors as they did after A.C.S. An increase in errors occurred in each of the three writing tasks: writing 'to command', dictation and copying. The increase

TABLE II

Written spelling errors in controls, in 21 patients in A.C.S. (examination A) and in the same patients out of A.C.S. (examination B). Comparison of the mean values

	Controls (10 *S*s)	During A.C.S. exam. A (21 *S*s)	Post A.C.S. exam. B (21 *S*s)	*t* (A/B)	*t* (B/C)
Total spelling errors	5.10	15.00	5.50	3.15***	0.42 Ns
Writing to command	1.10	10.45	1.90	1.71 Ns	0.51 Ns
Writing to dictation	8.00	19.83	9.98	3.94***	0.42 Ns
Copying	0.00	7.86	2.10	2.14*	0.99 Ns
Nouns	14.00	20.95	10.98	2.29*	0.37 Ns
Verbs	2.00	19.38	4.71	3.38***	1.00 Ns
Adjectives	5.00	15.62	7.38	1.74 Ns	0.54 Ns
Other words	0.00	5.12	1.10	2.88***	1.25 Ns

(1) Total percentage of misspelled words (expressed in per cent of the total number of written words).

(2) Percentage according to the writing task (in per cent of the number of words of the task considered).

(3) Percentage according to the grammatical form class (in per cent of the number of words from the considered form-class).

'Writing to command' means the composition by the patient of a sentence on a specific subject.

The A/B comparison was made by a *t*-test for paired data; the B/C comparison by a regular *t*-test. * $p < 0.05$; *** $p < 0.01$.

TABLE III

Analysis of the spelling errors in regard to the type of 'transformation', the involved element and the location of the error in the word (see text)

	Controls (C) (10 *Ss*)	During A.C.S. exam. A (21 *Ss*)	Post A.C.S. exam. B (21 *Ss*)	*t* (A/B)	*t* (B/C)
Omission	1.50	8.60	3.10	3.46***	0.75 Ns
Addition	1.10	5.43	3.74	0.81 Ns	1.08 Ns
Substitution	1.70	4.90	1.67	3.44***	0.02 Ns
Inversion	0.40	0.36	0.76	1.09 Ns	0.72 Ns
Consonants	1.90	10.50	3.52	3.01***	0.72 Ns
Vowels	2.30	4.74	2.48	1.63 Ns	0.10 Ns
Syllables	0.40	1.74	0.81	1.17 Ns	0.68 Ns
Whole words	0.20	3.36	0.62	3.48***	0.69 Ns
First 2 letters	0.00	0.97	0.33	1.06 Ns	Ns
Last 2 letters	0.90	5.97	1.85	2.56 *	1.49 Ns
Middle of word	1.10	7.87	4.79	1.69 Ns	1.26 Ns

Errors are expressed in percentages of the total number of written words.

The A B comparison was made by a *t*-test for paired data; the B C comparison by a regular *t*-test. *$p < 0.05$; ***$p < 0.01$.

in errors in writing 'to command' is not, however, statistically significant.

When we turn to the *grammatical category* of the misspelled word, we find an increase in spelling errors mainly in verbs and small grammatical words (prepositions; pronouns; articles), designated in the table as 'other words', and, to a lesser degree, nouns. Misspelling of verbs often consisted in the modification of auxiliary verbs or of the grammatical endings of regular verbs (e.g. -ed, -s). Small grammatical words were either omitted or substituted for by other words. On the whole, grammatical words and endings, though highly familiar, appeared to be very fragile. Difficulties in these categories are, of course, well known in some varieties of aphasics.

Errors were also analyzed according to the '*transformation*' leading from the target word to the word actually written. We adopted the following classification of misspelling: *omission* ('clar' for 'clear'), *addition* ('carefull' for 'careful'), *substitution* ('lan lin' for 'Van Line') and *inversion* ('librety' for 'liberty') (Lecours, 1967). Often, an erroneous word concontained more than one type of error. Such errors could involve isolated

letters, as well as groups of letters ('arearea' for 'area') and whole words ('if it is' for 'if he is'). The nature of the 'transformation', the element involved (consonant, vowel, syllable or word) and the location of the word were noted.

The data pertaining to this part of the study are reported in Table III. When we consider the *nature* of the 'transformations', we find that the increase of written misspelling during A.C.S. appears to be related to an increase in omission and substitution errors. Addition and inversion errors did not change significantly. There was, however, a trend to reduplication of the last letters of the word (e.g. 'falll', 'businesss'...) or to reduplication of whole words (e.g. 'if if he is'). An increase in omission errors is not surprising in a disorder the main feature of which is an attention defect. Less expected is the stability of inversion errors. Our patients are thus different from cases of developmental dysgraphia (Lecours, 1967).

The study of the transformed *elements* revealed a high degree of involvement of consonants and of whole words (generally small grammatical words). The high error score for consonants reflects particularly an increase in omissions, and to a lesser degree, other types of transformation. It is in striking contrast to the low error rate for vowels, a difference which remains even when we balance for the unequal distribution of vowels and consonants in the test material.

The *location* of the 'transformation' was studied only for errors in single letters in words having five letters or more. We computed the number of errors involving the two first letters, the two last letters and the middle of the word. As demonstrated in Table III, errors involving the last two letters increased significantly while errors in the other categories did not.

The written spelling errors of the subjects seen after recovery from A.C.S. (examination B), were similar in every respect to those of the control group (see Tables II and III); some minor spatial or motor errors were found in only 3 patients of group 1 after recovery.

Writing Disturbances in Relation to Disorders of Higher Cortical Functions (H.C.F.)

The dysgraphia demonstrated by our patients was far more severe and constant than any other concomitant disorder. In spoken language,

there were some word-finding difficulties and a few errors in reading aloud and repetition, but the overall pattern of spontaneous speech was normal. On the other hand, oral spelling did not change significantly between the two testing sessions, furthermore, although this was not formally studied, we had the impression that an acutely confused patient could spell a word aloud better than he could write it; and when a patient was asked to compose a word with block letters, he made fewer errors than when he wrote it (we gave him only the letters composing the word). In regard to the other H.C.F., there was impairment in the drawing tasks and minimal difficulty with right-left orientation, finger recognition and calculation.

During the A.C.S. (examination A), the rate of misspelled words covaried with the motor difficulties in writing ($r=0.39$; $d.f.=28$; $p<0.05$), as well as with disturbances in drawing[1] ($r=0.62$; $d.f.=28$; $p<0.001$). When the scores obtained out of A.C.S. (examination B) were considered, the disturbances on written spelling were correlated with the oral spelling score ($r=0.63$; $d.f.=23$; $p<0.01$), and to a lesser degree, with the educational background ($r=0.39$; $d.f.=23$; $p<0.10$).

III. DISCUSSION

These results indicate that a writing disorder may be associated with an A.C.S. regardless of the etiology. This writing disorder has common characteristics which vary only in severity and which involve the motor and the spatial aspect of writing, as well as spelling and syntax. it is a transient phenomenon which disappears when the A.C.S. has cleared. This dysgraphia is the most striking and the most constant linguistic disorder seen in A.C.S.

The spelling disorder has the following features: a high error rate in consonants and of small grammatical words in their entirety, a high rate of omission and substitution, a high rate of involvement of the last letters of the words.

The fact that a widespread acute cerebral disorder can lead to a relatively isolated dysgraphia seems, to us, to be the most interesting conclusion of this study and may throw a new light on the old problem of pure agraphia (P.A.).

We will review the cases which have been claimed to be examples of

P.A. We will not discuss here the 'P.A. cases' in which agraphia was associated with disorder of oral language (Eskridge and Parkhill, 1896), reading (Marcus, 1937), or of fine hand movements, (Mahoudeau *et al.*, 1951). The agraphia, in these cases, was part of a wider syndrome and cannot be considered 'pure'.

If we focus on the cases in which agraphia was the sole or the most prominent symptom, we are struck by the high proportion of reports in which mention is made of 'mental deterioration', 'obtundation', 'brain atrophy', 'increased intra-cranial pressure'. Thus, the patient of McConnell (1905) was said "to fix his attention and keep his mental machinery in action with difficulty". The patient of Sinico (1926) is reported as being in a state of 'demenziale progressivo'. In the first of Hécaen's series (in which the agraphia is not actually described as a 'symptome privilégié'), 4 out of 7 patients had brain tumors and were obtunded, 2 had cortical atrophy and 1 had been operated on for a frontal epileptogenic scar and was reported as both mentally deteriorated and illiterate (Hécaen and Angelergues, 1966). In Hécaen's second series, 4 patients out of 6 had intellectual impairments, a fifth patient was said to be alert but had a brain tumor; only the last patient, examined only one month after a cerebrovascular accident, was said not to be deteriorated or confused (Dubois *et al.*, 1969).

The famous case of Gordinier (1899) is, to us, no more conclusive: (1) there was an extensive frontal lobe tumor with signs of increased intracranial pressure (diplopia, papilledema); (2) the writing performances demonstrated by his patient (reiterated drawing of curves) might be considered as showing frontal perseveration rather than agraphia. We raise similar objections to the cases of Campbell (1911) and the first case of Mahoudeau (1950).

The case reported by Penfield and Roberts (1959) was a young man, alert preoperatively, who was operated on for a frontal epileptogenic scar and presented an agraphia on the 9th day after the operation. The fact that the agraphia was a delayed and transitory phenomenon at least raises the possibility of a confusional state on the basis of edema. The same problems arise in the case of Morselli (1930).

The case of Nielsen (1946) was a left-handed man who had always written with the right hand. He developed an agraphia of the right hand alone in association with a right hemiplegia affecting primarily the leg.

This clinical picture strongly suggests an anterior cerebral occlusion with probable infarction of the callosum in a patient with speech in the right hemisphere. Although we have much less information about the case of Pitres (1884), the same analysis may well apply. These cases thus resemble the case of Geschwind and Kaplan (1962) who manifested a left unilateral agraphia. This patient showed other signs of callosal disconnection and subsequent post-mortem confirmation of an infarction of the corpus callosum without any lesion in the right hemisphere.

In summary, most of the cases in the literature support the idea that an isolated disorder of writing occurs primarily in the presence of a diffuse brain dysfunction. We assume that, with the possible exceptions of one of Hécaen's cases and Henschen's (1922) personal cases, in which the lesions were vascular, the so called 'P.A. syndromes' were the expressions of general brain dysfunctions, not of specific language output disturbances.

Dubois *et al.* (1969) have called attention to a relative increase in P.A. in left handed subjects. The reasons for this phenomenon are not clear, and we will therefore not discuss this problem here. We wish to point out that our preceding comments may perhaps not apply to this group, since the possibility of pure agraphia in left-handers on the basis of a different cerebral organization cannot be ruled out at this time.

We do not imply that the dysgraphia found in A.C.S. patients is necessarily identical to the one described in cases of P.A.: (1) impairment of the spatial aspect of writing is more marked and more frequent in A.C.S. than in cases of P.A. These spatial disturbances are similar to those described by Marcie *et al.* (1965) in cases with right-sided tumors. If we assume that an A.C.S. results in a global dysfunction of the cerebrum, the spatial defect we have observed can indeed be accounted for by the right hemisphere involvement. (2) the written spelling disorder in A.C.S. is, on the whole less severe than the ones reported in cases of P.A., in Hécaen's last series for example. It is possible that a lesion of the left hemisphere might play a role, possibly through involvement of the cortical language areas, in 'magnifying' a writing disorder provided by a superadded diffuse brain dysfunction. We would wish, however, to point out that the relatively lower degree of written spelling disorder in our cases may not reflect a true difference but may be a result of patient selection. Since patients were excluded from the study unless they could

cooperate for the entire test battery, some more severely involved subjects were almost certainly omitted. We have, in fact, seen clinically patients in A.C.S. with even more severe writing disorders than those described here.

Writing thus appears to be a very delicate, fragile task. Such fragility has been pointed out in the past. Davis and Davis (1939), in an experimental study of hypoxemia in normal (1939), subjects, noted that handwriting changed simultaneously with the E.E.G. when the subjects were oxygen deprived; they stated that writing seemed to be "a very delicate indicator of consciousness impairment". Benton and Abramson (1952), in their studies of the Gerstmann syndrome in patients recovering from electro-convulsive therapy, mentioned the presence of dysgraphia but did not point out any discrepancy between this symptom and the other components of the syndrome.

Why is writing so fragile? It is possible that writing is readily disturbed because it depends on so many components (motor, praxic, visuospatial as well as kinetic and linguistic). Furthermore, most normal humans exercise their speaking abilities and their comprehension of spoken language constantly. Many people, although fewer, exercise their reading comprehension abilities very extensively. It is, however, only a minute fraction of the population, even among the highly educated, who use their writing abilities extensively. Writing is therefore very rarely, if ever, an overlearned and automatic skill.

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NOTES

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[1] Motor difficulties in writing and drawing difficulties were scored according to a three point scale by two independent judges.

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CHAPTER XXIV

A REVIEW*

1972

Traumatic Aphasia: Its Syndromes, Psychology and Treatment, by. A. R. Luria. Translated from the Russian by Douglas Bowen, with a foreword by MacDonald Critchley. *Janua Linguarum, series maior* **5** (1970), Mouton, The Hague.

Luria is one of the small group of investigators whose work has, in the period since the close of World War II, revived interest in aphasia, and he has consistently remained one of the principal contributors to this area. This book was first published in Russian in 1947, but has unfortunately not been available in English until now.[1] The publication of this work is specially important, since it is central to an understanding of L's later contributions.

Since the literature on aphasia is not well known, it is especially important to view Luria's contributions in the context of earlier work. Although most of the investigations on aphasia between 1860 and World War I were carried out by clinical neurologists, interest in it declined rapidly among this group after 1918, particularly in English-speaking countries. Although aphasia is a very common phenomenon in neurological practice, it has generally continued to be neglected even by academic neurologists. The reasons for the continued loss of interest included the feeling that the literature was highly confused, an impression furthered by the intemperate polemic of Head (1926). Furthermore, as biochemistry played an increasingly great role in medical research, the study of aphasia began to appear as an exotic and unscientific activity suitable only for those of philosophical inclinations. The new dynamic psychiatry created a hostile atmosphere for attempts to tie changes in the brain to behavior, and furthermore attracted away many who would have been primarily interested in the behavioral aspects of neurology. The results of this can still be seen today, so that even neurological textbooks may contain little or no discussion of aphasia.

Unfortunately, the decline in interest among neurologists was not

parallelled by an increased interest among psychologists. Indeed, for a long period, interest in aphasia was maintained in the United States primarily by the rapidly growing group of speech pathologists, who, however, showed little interest in the neurological aspects of the disorder.

Since World War II there has been a slow but definite increase in interest. Speech pathologists, psychologists, and neurologists are now working in this area, although the number of active contributors is still small when one considers the enormous medical and social importance of the problem, and its unique theoretical importance in giving clues to the neural foundations of language. Linguists have barely been concerned, and there are probably not a half-dozen who have had an intimate continued personal contact with aphasics.

One of the major continuing hindrances to growth in the field, which has undoubtedly kept out many who might wish to become involved more intimately, is the intense difficulty in reading the literature. Almost every author in the field represents himself as championing a totally new approach, with all its attendant philosophical impedimenta. Every writer appears to have his own classification, so that the unprepared reader is quite likely to conclude that the phenomena can be sliced up in an almost limitless number of ways. If he considers the relationship of brain lesions to aphasia, he is beset on the one hand by those who argue that almost no correlation exists, and on the other by those who argue pinpoint localization. It is no surprise if many scholars feel that the field of aphasia is most similar to Matthew Arnold's darkling plain, where ignorant armies clash by night.

The apparent lack of any common body of knowledge or tradition has perhaps been the greatest deterrent to advancement of this intriguing and important field. My contention is that the state of affairs in aphasia is in fact considerably less chaotic than appears at first glance. This will be apparent when it is realized that each new author is not as independent of the past as is often alleged, and that widely differing terminologies reflect considerably less disagreement about the facts than one might at first suppose.

I will therefore first try to place Luria in historical context. As soon as one sees more clearly the traditions he embodies and his areas of agreement with others, it will be easier to evaluate the magnitude of his contributions

and the areas of substantive disagreement in which new research efforts will be required.

It is probably best, in assessing any author in this field, to disregard his own explicitly stated philosophical presuppositions. The stated premises of most authors in aphasia seem to be little reflected in the actual content of their work. Thus L approves strongly of Jackson (1958), but so do all other writers on aphasia (except perhaps myself, who can approve only mildly). L is against isolated cortical 'centers' for advanced functions, a view shared by Wernicke (1874). L strongly stresses the adjectives 'dynamic', 'evolutionary', 'mobile', and 'voluntary' in description of functional systems, but also reveals himself as an admirer of Pavlov – who, if we are to believe Arthur Koestler, is the very embodiment of the 'mechanistic' approach to brain function. Furthermore, Konorski, who also is a follower of Pavlov, has stressed (1967) certain aspects of aphasia which are not even mentioned by L. This difficulty in placing writers on aphasia in context, based on their own stated philosophical presuppositions, is true of many other authors. It is best therefore to turn in each instance to substantive contributions.

Authors on aphasia can be divided into those who place importance on relating language disorders to the causative lesions within the nervous system, and those who believe that language disorders should be studied entirely as pure linguistic phenomena. L's position here is clear. Despite his concern for the phenomenology of linguistic and other disorders, he is uncompromising throughout in his stress on attempting to understand how these relate to the changed physiology of the brain. The significance of this point of view will become clearer in further discussion.

A second major division between students of aphasia is that which is sometimes described familiarly as the distinction between 'lumpers' and 'splitters'. The lumpers are those who either deny the existence of different aphasic syndromes, or are prepared to admit only the grossest of differences, such as that between 'expressive' and 'receptive' aphasias, an apparently simple differentiation accepted by many authors. The splitters are those who accept the existence of many different syndromes. Again L's position is clear: he is an outright splitter. There is a further differentiation among the splitters. There are those who are basically lumpers; i.e., they believe in the existence of only one basic type of aphasia, and therefore must explain away the existence of different syndromes. One explanation advanced is that the different syndromes are the result of damage to a supposed single speech area, with simultaneous damage to adjacent nonlanguage regions to account for special difficulties, e.g. in reading. Another explanation is that the different syndromes are merely different degrees of severity of a basic single form of aphasia. While L would deny the importance neither of neighborhood lesions nor of changes in symptomatology with variations in severity, his fundamental view as to the major cause of differences between syndromes is a very classical one: they are the result of localized damage to separate regions, each playing a specialized role in the physiology of language. In brief, L's position is essentially that of a classical localizationist; he is close to such works as Wernicke (1874) and Dejerine (1914), which represented the main line of the classical tradition, rather than to such as Kleist (1934, 1962), which took an extreme position as to the number of areas with differentiable functions. Unfortunately, there is a strong tendency to caricature the classical view as being 'phrenological'; thus it is generally not appreciated that Wernicke, the main founder of the classical tradition, was just as critical of the approach which assumed that a specific brain region could be assigned to each nameable psychological function as he was of the equally untenable view that the brain was an equipotential organ. Like

Wernicke and his followers, L stresses the existence of a small number of syndromes, and he puts great importance on the consistency of the syndromes produced by specific lesions.

L is presented with a problem in dealing with localization, since the type of case studied in this book offers many difficulties to an investigator concerned with correlating the clinical picture and the site of the lesion. The patients presented are cases who had suffered from penetrating brain wounds. Many readers, particularly those without a background in deseases of the nervous system, may be misled by the statement in Critchley's introduction that missiles entering the brain typically cause clean punched-out areas of damage, and therefore represent the ideal material for study of the effects of delimited cerebral damage. Unfortunately this statement is one that neither I nor many neurologists, neurosurgeons, or neuropathologists would accept. In the first place, the site of entry of the missile is no index of the direction in which it is moving, so that missiles entering at identical sites may end up in widely differing places. Second, in their passage through the brain they may produce shearing effects and damage blood vessels. Further damage may be done by infection of the wound or by the necessary surgical steps taken to repair it. It is no surprise, therefore, that penetrating injury is in fact much less likely to present a consistent picture than are the sequelae of occlusion of blood vessels. Furthermore, few cases of traumatically induced aphasia have come to post-mortem examination for verification of the loci of damage, while many cases secondary to primary vascular disease have been subjected to extensive anatomical study. Finally, the impression is often given that cases of vascular disease have widespread involvement of the brain, but again pathological study has shown many cases with isolated damage.

The inconsistency of traumatic material appears most strikingly in the famous diagram in Conrad (1954), often advanced as an argument against any very precise localization of the aphasias. Conrad's diagram shows the sites of entry of missile wounds leading to aphasia, but a very large proportion of these entry wounds lie over regions in which an isolated lesion has never been found in any case of aphasia examined at post-mortem. L, however, brilliantly circumvents the difficulties of dealing with this type of material by the use of a more sophisticated approach than that of Conrad, and indeed with very different conclusions.

He avoids the error of simply mapping the location of wounds producing a particular syndrome, but instead gives the *percentage* of all wounds in each region which produce that syndrome. It is necessary to use percentages rather than absolute figures, since penetrating brain wounds probably do not occur with equal frequency in all parts of the brain.

L adds another particularly important step to this analysis, i.e. the documentation not only of the number of cases showing a particular syndrome in a given region in the early period after injury, but also the percentage of cases at each location which show more persistent syndromes over time. This is particularly important in further eliminating the effects of such factors as swelling of the brain, which typically occur in the early stages after injury. By the use of these careful methods of analysis, L has considerably overcome many of the disadvantages of material based on penetrating injuries of the brain.

We can now turn to L's classification of aphasia. His terminology is his own, and we must therefore consider carefully whether he has indeed classified his material in an entirely different way from previous authors, or whether he has merely coined a new set of names. If we read the descriptions of his cases, it is clear that most of the syndromes he describes are readily assimilated to the classical varieties. L speaks of 'acoustic aphasia' (120) which he specifically equates to Wernicke's (or sensory) aphasia, i.e. the syndrome of lack of auditory comprehension and inability to repeat, associated with abnormal speech production but without difficulty in articulation. L's syndrome of 'acousto-mnestic aphasia' (136) appears to resemble anomic aphasia and to result from posterior basal lesions.

The syndrome of 'afferent (apraxic) motor aphasia' (149) arises from lesions in the lower portion of the post-central gyrus. L's description appears to put great emphasis on the substitution of phonemes in the speech of these patients. This tendency to 'literal' (or phonemic) paraphasia is characteristic of the syndrome of conduction aphasia as described by others, although L does not put the same stress on disorders of repetition in this syndrome that other authors do (such as Goldstein, 1927, or Konorski, 1967). 'Efferent (kinetic) motor aphasia' (185), which results from lesions in Broca's area, i.e. the portion of the cortex lying just forward of the motor region for the face, tongue, and lips, and adjacent parts of the frontal lobe, is characterized by grammatically simplified stumbling speech, a picture generally compatible with the classic Broca's aphasia. L describes still another syndrome, that of 'frontal dynamic aphasia' (199), which results from lesions lying forward of Broca's area. These patients show a picture which corresponds to the 'motor transcortical aphasia' of Goldstein (1917), characterized by lack of spontaneous speech, but preserved repetition.

Certain syndromes do not appear in L's book; their absence is a reflection of the kind of pathological lesion with which he is dealing here. Since he is primarily considering wounds produced by missiles, some of the clinical pictures which cannot be produced in this way, but which can result from occlusion of blood vessels, are absent. Thus the syndromes of the corpus callosum (cf. Geschwind, 1965), which is rarely damaged in cases of penetrating trauma to the brain, are not mentioned. Also omitted is the syn-

drome of pure alexia without agraphia (Geschwind and Fusillo, 1966), produced by occlusion of the left posterior cerebral artery, with resultant destruction of the left visual cortex and the posterior end of the corpus callosum. Note also pure word-deafness (Geschwind, 1965), a syndrome which requires a lesion deep in the temporal lobe with sparing of the overlying cortex; this obviously cannot be produced by a penetrating missile, but can be produced by vascular disease. The same is true for the syndrome of the 'isolated speech area', in which the speech regions are intact, but cut off from connections to other cortical regions (Geschwind *et al.*, 1968), manifested by the unusual combination of severe loss of auditory comprehension but intact repetition.

It is perhaps the concentration on cortical lesions which accounts for one feature which is lacking from L's analyses. Despite his strong anatomical orientation, he generally neglects the fiber connections (which run beneath the cortex) of the various cortical speech regions, and does not consider the syndromes which might result from disconnection of these regions. Other authors (Wernicke, Dejerine, and more recently Konorski and Geschwind, 1965, 1970), have put great emphasis on the importance of these in the cerebral organization of language.

I have spent considerable space in placing L's work in context. The conclusions drawn above should not be misconstrued. The fact of the basic agreement between L and earlier workers should not be taken to suggest that his work lacks originality or imagination; he is indeed one of the modern giants in this field. The existence of basic concordance between his work and that of the founders of the field only makes his new contributions more valuable. No field can advance without some elementary agreement as to basic findings.

Indeed, some of L's original contributions, as expressed in his other writings over the past twenty years, have become such integral components of thought in this area, and have appeared so frequently in the writings of others – who often fail to realize their origin – that we are likely to underestimate the stature of this book. At the time of its publication in 1947, it was undoubtedly the most important work on aphasia since the close of the classical period in the 1920's. Had it been translated into English at the time, it might have hastened the modern revival of interest in this area in France, Germany, and the English-speaking countries, where knowledge of Russian and even sensitivity to the possibility of important Soviet contributions were unfortunately generally lacking.

As mentioned earlier, L not only circumvented the difficulties of using cases of penetrating missile wounds in order to obtain localizing information, but also used the evolution of the syndromes over time so as to separate areas essential to language from those of secondary importance.

In fact, L's work contains perhaps the largest series of cases in which careful correlation of clinical data and anatomical localization has been carried out. Some of his conclusions bear particular weight because of the large number of cases. Thus he points out that, with lesions in the principal speech areas (i.e. those lying along the Sylvian fissure), total recovery is exceptional. Furthermore, slight forms of aphasia almost never occur with injury to these areas. This would seem to indicate that, although compensation does occur in some of these cases, most adult human brains possess the capacity for such compensation to a much more limited extent than is the case in the child with acquired aphasia. However, L puts great importance on the small number of cases in which penetrating injury of the principal zones produces no impairment, or in which recovery is rapid and complete. Because of his large number of cases, he is able to support the view that the degree of lateralization of speech functions varies between individuals, i.e. that dominance varies along a spectrum. This view had in fact been expressed frequently by older authors, but few of them had so much case material on which strongly to support this conclusion. L further points out that such a variation in the degree of recovery occurs even among patients who are apparently fully right-handed. However, mild forms of aphasia after penetrating wounds of the primary areas were seen only in patients who were themselves not right-handed – or, if right-handed, had a history of left-handers among their relatives. Furthermore, aphasia with right hemisphere lesions in right-handers occurred only when there was a familial history of left-handedness, and in many of these there was excellent recovery. In addition, left-handers rarely develop a severe permanent aphasia after severe damage to the right hemisphere. L's research anticipated later work which has shown repeatedly that the old doctrine that left-handers have primary speech representation in the right hemisphere is a much too simple view of the situation.

Let us now turn to another field in which L was a pioneer, that of the methodical investigation of the linguistic disorders in aphasia. It should be kept clearly in mind that, from the earliest days of the modern study of the aphasias in the 1860's, the criterion for the existence of aphasia has always been failure of linguistic expression or comprehension (although the disorder might be confined to a single modality, e.g. comprehension of written language). Wernicke had made the first great advance in

linguistic analysis by calling attention to the almost complementary differences in the linguistic disorders associated with Broca's and Wernicke's aphasias. In the former, the speech output is slow, effortful, with distorted phonemes, and 'agrammatism' (i.e. a lack of the small grammatical words such as *the*, *is*, and *if*, and a loss of endings); in the latter, speech is fluent, with production of normal phonemes, and an essentially normal grammatical skeleton, but with marked difficulty in word choice, the presence of empty words (such as *thing*), circumlocutions, incorrect word choices, substitution of phonemically correct sounds, and even frank neologisms. The study of linguistic errors was furthered by such works as Pick (1913), Bonhoeffer (1923), Salomon (1914), and Isserlin (1936). But L deserves the credit for introducing the experimental study of linguistic error, which has been continued in recent years by such works as Goodglass *et al.* (1970), Dubois *et al.* (1964), and Green (1969). This field is still in its infancy, for the reason that the linguistic breakdown in aphasia must be studied as a special problem: the deterioration of language does not obey in a simple manner the rules of any linguistic theory. E.g., even in patients with severe comprehension disturbance, the understanding of commands for whole-body movements (such as *Stand up*, *Turn around*, *Stand at attention*) may be remarkably preserved, while understanding of commands for individual movements of the limbs of face (e.g., *Make a fist, Put out your tongue*) may be lost, a distinction probably related to differences in the anatomical organization of the two types of movement. On the other hand, one does not see selective disorders of such well-accepted linguistic categories as adjectives or adverbs. However, while these facts do not support, let us say, the current transformational theories, neither do they necessarily argue against them. I would have to accept the view that what one might call the realization in hardware of an axiomatic system would not necessarily be, so to speak, isomorphic with that system. One would hardly expect that damage to an ideal computer, capable of deriving theorems of Euclidean geometry, would produce loss of individual axioms. On the other hand, one should also not at this time accept the view that the breakdown in aphasia necessarily casts no light on linguistic theory. My personal view would be that the knowledge derived in the future from aphasia will have an important impact on the fundamental concepts of linguistics, but this remains to be proven.

An important series of experiments reported by L relate to cases of 'acoustic temporal aphasia' (Wernicke's aphasia in classical terms), in which he regards the essential disorder as being impairment of phoneme discrimination. L claims (115) that, in general, these patients fail in discriminations between phonemes differing in a single distinctive feature ('oppositional' phonemes), but perform well with phonemes differing in several features ('disjunctive' phonemes), while aphasics with lesions elsewhere do not show this marked impairment. This concept is a challenging and important one, and the question of the full validity of this interpretation remains open. There appear to be patients with the typical syndrome of temporal aphasia whose failure appears to be much more at the semantic than the phonemic level. L admits the existence of such patients, and argues that their lesion is different from the one producing phonemic discrimination disorder. In any case, further study is needed of this important problem.

A more serious problem results from the analysis of what L regards as 'secondary' disorders in these patients, e.g. disturbance of spoken speech or reading comprehension. It would be difficult to attribute these directly to failure of phonemic hearing, since patients with so-called 'pure-word deafness' may have an even more severe disorder of this kind, but speak and read normally.

A similar difficulty arises in relation to L's interpretation of what he calls 'afferent (apraxic) aphasia', with lesions in the parietal lobe near the Sylvian fissure. L argues that lack of afferent feedback is important, and that this results in the breakdown of differentiations among similar articulatory positions, so that phonemic errors are made. It is true that these patients frequently make phoneme substitutions (so-called 'literal' paraphasias); but the phonemes are themselves generally correct, thus suggesting that it is choice among correct articulatory patterns which is at fault. Furthermore, these patients also often make errors at the semantic rather than the phonemic level. In addition, the special difficulty that these patients show in repetition of the small grammatical words suggests a disorder more extensive than simple involvement of articulatory schemes. This too is an area that deserves further study to follow-up L's pioneering observations.

In his discussion of 'efferent motor aphasia' (the classical Broca's aphasia), L points out (196) that the 'breakdown of grammatical speech structure into single nominative words is not simply the result of an articulatory difficulty or due to a disturbance of the "conservation of energy" to which many authors have referred in analysing the phenomenon of "telegraph style"... This is seen in the fact that, even though the patient attempts to correct the defects of his grammatical sentences, he is unable to do so.' I would agree fully with this observation, which stands in contradiction to the views of others who would argue that this form of aphasia is characterized only by breakdown at the level of articulatory schemata.

The advance in linguistics in recent years at both the theoretical and descriptive levels, along with the increasing development of new technical methodologies such as those employed by the workers at the Haskins Laboratory, as well as dichotic listening techniques (cf. Shankweiler and Studdert-Kennedy, 1967), promise to lead to a greatly increased number of experimental studies of linguistic changes in the aphasic. L, however, will stand as one of the pioneers of the modern development of this area.

I will not discuss here in detail some of the other topics in the book to which L assigns considerable space. There is a long section allotted to methods of examination for particular deficits; this is invaluable for those who wish to carry on methodical testing of patients. It deserves comment that this section reflects L's imaginative and experimental approach. He has constantly devised new testing techniques, and has not fallen

prey to the all too frequent tendency to use a 'standard' battery of tests. While such standard batteries are useful in any unit for routine testing and for monitoring changes over time, they represent a constant danger if the investigator fails to bring out distinctions whose importance was not apparent to the original designers of the tests, or fails to discover new phenomena.

L devotes considerable discussion to his methods of therapy. I do not feel competent enough in this area to assess his methods in contrast to others advanced in the literature. Unfortunately, he does not provide us with series of patients treated by different techniques, so it is difficult to evaluate the effectiveness of his own approaches as against those of others. In any case, this area is still an important frontier for further research, since the patient impaired by permanent significant aphasia remains a challenge. With an increased knowledge of the mechanisms of these disorders at the linguistic, psychological, anatomical, and physiological levels, the possibility of rational experimental therapies, whether behavioral, surgical, or pharmacological, increases steadily. Luria stands as one of those who has contributed importantly at each of these levels of analysis.

NOTES

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[1] The present translation is based on a revision of 1959.

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CHAPTER XXV

CONDUCTION APHASIA

A Clinicopathological Study

1973

ABSTRACT. Three patients with conduction aphasia are described; in addition to the distinctive language disorder, two of them had severe ideomotor apraxia while the other was entirely free of apraxia. Postmortem examination revealed that the two patients with apraxia had entirely suprasylvian lesions involving the white matter deep to the parietal operculum, while the nonapraxic subject had suffered total destruction of the Wernicke area. Based on these findings and a review of the literature, we would propose the following: (1) Conduction aphasia is a distinct clinical syndrome, readily separable from other varieties of aphasia. (2) Conduction aphasia can result from a pure suprasylvian or a pure subsylvian lesion as well as from a combination of the two. (3) The presence of ideomotor apraxia in conduction aphasia implicates suprasylvian pathology.

Conduction aphasia was originally proposed as a symptomatically distinct form of aphasia by Wernicke (1874). The disorder is characterized by fluency and paraphasia in spontaneous speech with normal or nearly normal comprehension of spoken language, but markedly impaired repetition of words and phrases, usually with difficulty in naming. Apraxia of face and limb movement is a common but not invariable finding, and some of the patients have other abnormalities related to parietal lobe damage. Although conduction aphasia has been described by a number of authors (Lichtheim, 1885; Pötzl, 1924; Stengel and Lodge-Patch, 1955), some consider it rare (Goldstein, 1948; Luria, 1966), and it is not mentioned at all by others (Weisenburg and McBride, 1964; Nielsen, 1936; Brain, 1961). The disorder is easily defined because of its distinctive clinical picture and among the patients at the Boston Veterans Administration Hospital conduction aphasia constitutes between 5% and 10% of all new aphasic admissions. The conditions of most of these patients improve, returning to near-normal levels and therefore clinical pathological correlation has been infrequent. The availability of three postmortem studies on patients whose conditions were clinically diagnosed as conduction aphasia has prompted this report.

Wernicke (1874) hypothesized that Broca's area and the posterior temporal auditory association area were connected by a pathway running

through the insula, a lesion of which would give rise to a distinctive syndrome, *Leitungsaphasie* (conduction aphasia). Later Wernicke (1908) appeared to agree with von Monakow that the arcuate fasciculus, which runs through the parietal operculum, was a major link between the two areas. The clinical syndrome originally put forward by Wernicke did not include the feature of disordered repetition. This was added by later observers as a cardinal sign, so that the term 'repetition aphasia' has sometimes been used as a synonym for conduction aphasia. Goldstein (1911) used the term central aphasia to describe a syndrome with fluent paraphasic speech, normal or near-normal comprehension, and marked difficulty in repetition which he specifically equated to the syndrome of conduction aphasia. Unfortunately, other investigators (Brain, 1961 and Russell and Espir, 1961) have borrowed Goldstein's term, 'central aphasia', but use it in a different sense, since their description includes comprehension difficulty and does not stress disorder of repetition. Because of this inconsistent use of the term, we present the following criteria for the diagnosis of conduction aphasia:

(1) Fluent, paraphasic conversational speech. The paraphasia is most often of the literal variety (phonemic substitutions), but both verbal and neologistic paraphasias also occur. As a rule, the quantity of paraphasia is greatest acutely and decreases during stages of recovery.

(2) Normal comprehension. There patients exhibit no significant difficulty in the comprehension of normal conversation.

(3) Repetition disturbance of a significant degree. This disability may be at the single-word level but more often is demonstrated in repetition of phrases, short sentences, or multisyllabic words. Repetition may be contaminated by paraphasia (often self-recognized but usually unsuccessfully corrected), by a paraphrase of the target phrase or sentence, or by the failure to produce meaningful speech when attempting to repeat. At times, by paraphrasing, the patient can present information identical to that contained in the target sentence even though the words of the original sentence cannot be reproduced. In many cases the repetition of small grammatical words is particularly impaired compared to other categories while repetition of numbers is sometimes better.

The above findings *must* be present for the diagnosis of conduction aphasia. Several other signs occur sufficiently often to be worthy of comment. These include the following:

(1) Naming disturbance. Almost always present to some degree, naming troubles may vary from literal paraphasic contamination of one or more syllables of the desired word to a total inability to produce the appropriate word, even when phonetic cues are offered by the examiner.

(2) Reading disturbance. Characteristically, the patient with conduction aphasia cannot read aloud (because of paraphasic contamination) but can comprehend much or all that he reads. Some cases, however, do have an impairment in reading comprehension.

(3) Writing disturbance. This is always present but may vary in intensity from a mild spelling difficulty with occasional omission or reversal of letters to a profound agraphia.

(4) Bilateral ideomotor apraxia. This finding may be severe in some patients, insignificant or absent in others. The apraxia occurs primarily with buccofacial and limb activities while eye movements and whole body activities are usually performed normally.

(5) Elementary neurological abnormalities. These may be totally absent. A hemiparesis on the right may be present, but often there is a cortical sensory loss on the right. In several patients we have found contralateral diminution of pain sensitivity plus spontaneous pain similar to that of the thalamic syndrome. The lesions in these cases with changes in pain sensation may well be similar to the parietal operculum lesions described by Denny-Brown and Chambers (1958) and Biemond (1956).

Most of our patients with conduction aphasia have returned home and many have returned to their former work. Some have suffered additional cerebral infarcts at later times, clouding the clinical and the pathological pictures, and very few cases have come to postmortem study. The Table reviews the literature of the limited pathological material available for correlative study of conduction aphasia.

I. REPORT OF CASES

Case 1

A 73-year-old man was hospitalized in May of 1964. His difficulties had begun acutely six weeks previously when he had dropped a pen that he had been using and shortly thereafter noticed speech disturbance. A tremor of the right hand disappeared in several days. The persistence of

speech problems eventually brought him to the Aphasia Research Section for evaluation. He was right-handed, had a high school education, and had worked all of his life as a self-employed plumber. Past medical history was non-contributory.

Physical examination revealed an elderly white man in no distress. Blood pressure was 180/120 mm Hg, pulse rate 76 beats per minute and irregular. There were arthritic deformities of the interphalangeal joints and of the left knee. The chest had increased anteroposterior diameter, and the heart had an irregularly irregular rhythm. Otherwise, his physical condition was good.

He was alert, cooperative, well-oriented, and by indirect testing demonstrated normal memory function. Expressive language (described later) was abnormal. Cranial nerve examination showed only right visual field defect, most pronounced in the upper field but was also demonstrable in the lower field. Right facial paresis was so mild as to be questionable, and hearing was intact. Motor strength was within normal limits bilaterally; there were no changes in tone, no asymmetry in reflexes, and no difficulty in coordination. Sensory testing revealed a mild and inconsistent right-sided abnormality, particularly in position sense and on double simultaneous tactile stimulation.

Testing of language and related cortical functions revealed fluent conversational speech marred by frequent pauses for word-findings. Despite the many pauses, he pronounced many three-to-five-word phrases with normal inflection and rhythm. He showed more ease in using filler or relational words than substantive words. There were many paraphasias, both literal and verbal. He was aware of many of these substitutions and at times tried to correct himself, although these efforts almost invariably led to frustration and worsening of his expressive ability. If he allowed himself to carry on, ignoring errors, he could express most of his desires in an understandable manner. Multiple tests of comprehension of spoken language did not reveal any specific disturbance. Understanding of words, phrases, and long complex sentences were within normal limits when he was allowed to use a simple response such as 'yes' or 'no' or pointing. However, if he was asked to carry out a command, he either failed or demonstrated an extremely long latency; it was concluded that this was based on apraxia as it could be demonstrated that he understood the command completely. In contrast to his

excellent comprehension, his ability to repeat was profoundly disturbed. He could repeat single digits and some single syllable words, but with double digits, multisyllabic words, phrases, or sentences, paraphasic substitution grossly impaired the repetition. There was a disturbance of word-finding, easily demonstrated by asking for the names of objects, body parts, or colors.

He had difficulty reading aloud, due to paraphasic contamination. Comprehension of written material was better, and he regularly read a newspaper. Writing was disturbed. He could write a few words to dictation, occasionally signed his name and address, but could not write a sentence that he formulated himself. Right-left orientation and finger recognition were normal. On praxis testing he often failed even such simple buccofacial activities such as sticking out the tongue, coughing, or whistling. Once when asked to protrude his tongue, he succeeded after 120 seconds of continuous effort, smiled and said, "I knew you were going to ask me to do that, I've been practicing." The command was given only once and during the two minutes, he continuously grimaced and made other movements demonstrating ongoing attempts to carry out the command. Within 30 seconds of the successful tongue protrusion, he spontaneously and effortlessly licked his lips. Similar praxis difficulties were noted in limb activities, but whole body activities (standing up, turning around, walking backward, etc) were performed easily.

With speech therapy he improved in expressive ability but showed little improvement in repetition. He was discharged three months after admission and was seen regularly for several years. During this time he improved slightly in expressive ability, but his repetition problems persisted.

Three years later he developed a pronounced left paralysis, left conjugate gaze defect, and a semicoma. Improvement was too limited to allow careful language evaluation, and he died seven months later.

At postmortem examination, the brain weight (fresh specimen) was 1250 gm. There was moderate to severe atherosclerosis of the circle of Willis. A large, comparatively recent infarct in the right hemisphere involved the lower part of the second frontal convolution, the entire third frontal convolution, the first and second temporal gyri, the lower part of the precentral motor cortex, and the lower third of the postcentral ascending parietal gyrus. The lesion also affected part of the supramarginal

Reports of Conduction Aphasia With Autopsy

Author	Spontaneous Speech	Comprehension	Repetition	Naming
Lichtheim (1885)	Fluent, paraphasic	Normal	Poor	Slightly better than repetition
Pershing (1900)	Jargon	Good	Severely disturbed	Disturbed
Liepmann and Pappenheim (1914)	Fluent, Paraphasic	Good	Poor, much literal paraphasia	...
Pötzl (1924)	Essentially normal, occasional paraphasia, spoke both Czech and German	After short period, good	Severely disturbed	Disturbed
Stengel (1933)	Fluent, paraphasic, paragrammatic	Good	Severe ("worst function")	Word-finding pauses
Goldstein and Marmor (1938)	Fluent, no paraphasia	Good but not perfect	Severely disturbed, "total failure"	Severely disturbed
Coenen (1941) Kleist (1962)	Fluent, paraphasic	Good	Nil	Nil
Stengel and Lodge-Patch (1955)	Fluent, paraphasic	Fair	Nil	Nil
Hécaen *et al.* (1955) de Ajuriaguerra and Hécaen (1956)	Grammatical defect	Almost totally normal	Severely disturbed	Disturbed
Hoeft (1957)	Fluent, paraphasic, paragrammatic	Normal	Poor with much literal paraphasia	Disturbed
Kleist (1962)	...	Little disturbance	Severe	Nil
Kleist (1962)	Disturbed, much paraphasia	Mildly disturbed	Severely disturbed	...

ng	Writing	Praxis	Locus of Abnormality
, r; prehension, d	Poor	...	Perisylvian and insular softening with involvement of frontal opercula, very little of parietal or temporal opercula
, prehension,	Nil	...	Softening supramarginal gyrus going 2.5 cm deep
ud, r; prehension d	Poor	...	Left-parietal junction, temporal part of "Wernicke area", and deeper structures
turbed	Disturbed	Mild, not described	Left supramarginal circling back end of sylvian fissure
, r; prehension mal	Poor	"Normal"	Left first and second temporal gyri and angular gyrus – superficial and deep (postoperative intracerebral hematoma)
, prehension,	Nil	...	Large postsylvian infarct destroying first temporal, insula, lower supramarginal and angular gyri going deep to internal capsule
, r; prehension, er	Poor	Normal	Junction-temporal-parietal-second temporal, inferior angular and supramarginal gyri, cortical and deep
,	Nil	Nil	Left supramarginal cortex, deep lesion involving supramarginal and temporoparietal junction
, urbed	Disturbed	Ideational apraxia	Gross destruction of temporal lobe except for cortex of first temporal gyri; also supramarginal partially destroyed, especially deep; angular gyrus totally destroyed
, aphasic; prehension, d	Poor	Disturbed, "ideokinetic"	Severe involvement of Wernicke area, deep to temporoparietal junction
, d; prehension, d	...	...	First and second temporal convolutions, posterior supramarginal
	...	Ideational apraxia	Junction-temporoparietal, involving first and second transverse temporal gyri and supramarginal gyrus

gyrus and a small amount of the third temporal gyrus. The area of infarction went from the pial membrane overlying the cortex to the vicinity of the ventricle, undercutting the anterior half of the internal capsule and destroying the caudate nucleus, the putamen, and the insula. There was another large, well-healed infarct in the right cerebellar hemisphere.

In the left hemisphere there were older infarcts (Figure 1). Broca's area was spared, but there was a small subcortical infarction immediately in front of the rolandic fissure involving only the opercular part of the motor cortex. An infarct of the calcarine fissure on the left affected not only the lower lip of the sulcus but also extended forward to involve the lower third of the splenium of the corpus callosum. Another infarct affected the white matter of the supramarginal gyrus and had destroyed the overlying cortex. This lesion involved the supramarginal gyrus from the superior lip of the sylvian fissure to the vicinity of the intraparietal sulcus and extended in depth from the convexity of the hemisphere to within 1 cm of the ventricular wall. Further caudally there was an extension of degenerating subcortical white matter underlying parts of the angular and superior parietal gyrus.

Case 2

A 36-year-old active-duty army sergeant was transferred to the Boston Veterans Administration Hospital on July 22, 1970. He spoke only English and had earned a high school diploma in the army where he had served for 19 years. He was right-handed but did have one daughter who was strongly left-handed.

In 1960 hypertension had been diagnosed, and in 1965 (age 31) he had suffered a myocardial infarction. Subsequently he suffered from coronary insufficiency and had remained hypertensive He had been treated with digoxin.

The present illness started with a momentary lapse of consciousness while driving. Approximately half an hour later he had a similar episode and hit a car. He was taken to a nearby army hospital where examination revealed aphasia, hemiparesis on the right, and congestive heart failure. Results of lumbar puncture were reported as normal. He was treated with diuretics and bed rest and in six days lost 8.58 kg (19 lb) with considerable improvement in general condition. Neurologic examination noted fluent,

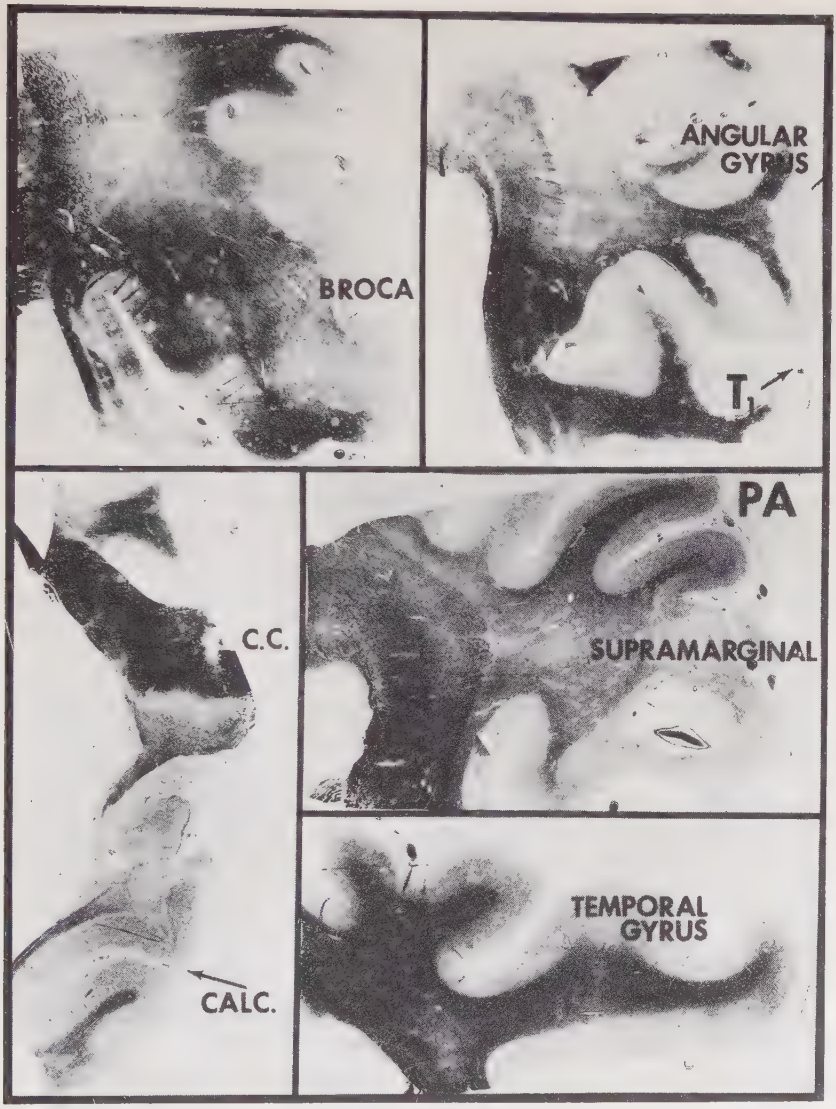

Fig. 1. Detail of left hemisphere lesions stained with Kluver-Barrera myelin stain (case 1). Small subcortical infarct in Broca area (top left), infarct of calcarine fissure and lower one third of corpus callosum (bottom left). Underlying white matter of angular and supramarginal gyri is severely affected by infarct. Temporal gyrus is normal.

paraphasic speech, good comprehension, and severe repetition disturbance. There was hypesthesia of the right hand only and minimal weakness of the right upper and lower extremities. Three weeks later he was transferred to Boston.

On admission his blood pressure was 130 80 mm Hg, the heart rate 110 beats per minute with multiple ectopic beats. His general physical condition was excellent, and until the day of his death, there were no significant general physical disturbances. Admission neurological examination revealed no abnormality of cranial nerves; in particular, the visual fields were full to both confrontation and tangent screen testing and opticokinetic nystagmus was normal bilaterally. There was a minimal, clinically insignificant right-sided weakness. He was fully ambulatory, coordination was excellent, the reflexes were brisk and equal, and the plantar responses were flexor. There was slight diminution of pain, touch, and temperature on the right side of the face and right arm and mild astereognosis of the right hand, but position sense, graphesthesia, and two-point discrimination were intact bilaterally.

Mental status examination revealed a bright, alert, cooperative patient who was fully oriented. His affect was consistently one of mild euphoria, and he was always cooperative in both testing and therapy.

Testing of language and related cortical functions revealed fluent and effortless conversational speech, free of dysarthria, but contaminated by many literal and verbal paraphasias. He was aware of only a portion of these errors, and when he attempted self-correction he usually failed. Word-finding difficulties were noted. Series speech (counting and reciting the days and months) was performed without difficulty. In singing he produced a good melody, but the lyrics were contaminated with considerable jargon. Comprehension was tested repeatedly, by many examiners, and was always found normal. These evaluations included tests utilizing abstract information, tests dependent on unusual grammatical form, and tests of sequential pointing. He easily identified nonverbal sounds in tests for auditory agnosia. In contrast, he had severe difficulty in the repetition of spoken language, even of single words. Thus, when asked to say "blue," he said, "f-f-f-ee – I can't do it." When asked to say "rifle," he said, "rickle - rif-fle – gun." Repetition of digits and letters was also poor, although they were handled better than words; many paraphasic errors were made when he attempted to repeat nonsense material or

foreign phrases. Naming was disturbed, most of the errors being caused by literal paraphasic contamination, often so excessive as to be considered neologistic. Naming was not helped by phonetic cuing, but he occasionally responded correctly to contextual cues. There were similar difficulties when he attempted to name colors or body parts. In contrast, he could always point to the correct object or color when the name was given.

Attempts to read aloud always produced considerable paraphasic contamination. Thus, when asked to read aloud the phrase, "Stick out your tongue," he said, "Snick out your toe." He had much less difficulty comprehending written material, and he could carry out a written command or answer a written question even though unable to read it aloud. He could write numbers and individual letters to dictation and when asked to copy words made few errors. However, if words were dictated, only a few letters were written and these were usually incorrect. Similarly, he could not spell out loud, although he readily recognized spelled words. He had no difficulty recognizing finger names, and he could differentiate right and left on himself and the examiner without difficulty. When verbal commands were given for buccofacial, limb, or whole body activities, he immediately and accurately carried these out. At no time was any significant degree of ideomotor apraxia demonstrated.

His hospital course was one of steady improvement. During this time his spontaneous speech became quite understandable, and he communicated freely with his fellow patients. He continued to have the same difficulties in reading aloud, writing, and repetition, and his ability to name was still disturbed by phonemic substitutions. Three months after admission he collapsed and died immediately.

The brain weight (fresh) was 1400 gm. Inspection of the hemispheres showed a 4 × 1-cm area of cortical softening beginning 2 to 3 cm caudal to the onset of the horizontal portion of the sylvian fissure and progressing caudally for about 4 cm, affecting the posterior half of the left superior temporal gyrus and reaching to the vicinity of the angular gyrus. This lesion appeared to be an old, somewhat sunken, yellowish area of infarction.

Coronal sections showed a tiny area of infarction in the middle third of the left claustrum. About 1 cm further caudally the large infarct extended to involve both lips of the sylvian fissure, i.e., the lowest aspect of the supramarginal gyrus, the insula, and the upper aspect of the planum temporale, sparing the Heschl gyrus but completely destroying

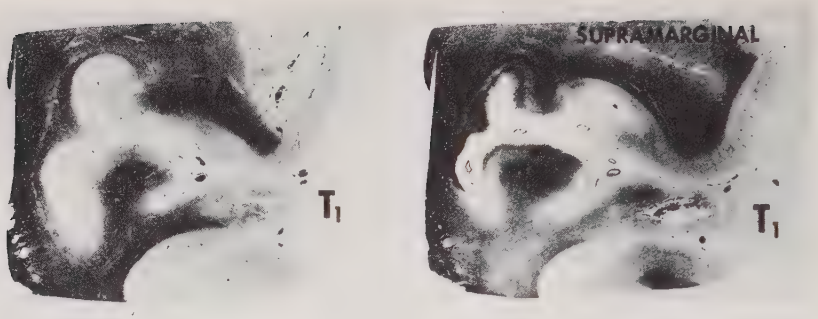

Fig. 2. Details of left hemisphere lesions with Kluver-Barrera myelin stain (case 2). Heschl gyrus (H) is intact but underlying white matter is severely affected. Posterior third of first temporal gyrus (T_1) and cortical ribbon of operculum (PA) are infarcted.

the posterior half of the first temporal gyrus. The zone of infarction extended caudally to the temporo-occipital junction, sparing the angular gyrus. The lesion involved the entire cortex and subcortical white matter of the Wernicke area. The rest of the brain was unremarkable.

Blocks to include both lips of the sylvian fissure of the left hemisphere were taken and embedded in paraffin. Serial sections were stained with hematoxylin-eosin, cresyl violet, and luxol fast blue stains (Figure 2). Microscopically the lesions were all consistent with old infarctions, well-healed, but the extent and severity of tissue destruction varied along the cortex lining the sylvian valley. The cortex of the post-central ascending parietal gyrus showed a discreet partial laminar necrosis expressed by spongy change, mild capillary proliferation, and ischemic cellular changes. Deep in the insula the lesion changed to linear necrosis along the outer granular layer and separated from the pia by a barrier of proliferating astrocytes. About two thirds of the Heschl gyrus was spared, but the extent of the necrosis suggested that the preserved portion was isolated from the rest. The portion of white matter surrounding the tip of the lenticular nucleus and deep supramarginal gyrus (i.e., the area pierced by fibers of the fasciculus arcuatus) was normal in appearance and no lesions could be detected in them.

Case 3

A 63-year-old right-handed retired carpenter entered the Boston City

Hospital because of speech difficulty and changes in behavior. There was a five-year history of treated hypertension. Fourteen months before his admission he had had a generalized seizure. At that time the left carotid pulse was decreased, but neurological examination revealed no evidence of aphasia, agnosia, alexia, agraphia, or difficulty with right-left orientation. Results of studies including chemical analyses of the blood, skull x-ray films, electro-encephalogram, brain scan, lumbar puncture, and pneumoencephalography showed only a mildly elevated blood glucose level and a cerebrospinal fluid (CSF) protein level of 88 mg/100 ml. Three months prior to admission, he suddenly became unable to speak coherently and had difficulty finding words. When questioned about this problem, the patient answered: "I know what I want to say, but the right word just does not come out". Comprehension for normal conversation was intact. One month before admission he was found wandering about the house during the middle of the night. Thereafter members of his family said that he "...got lost all the time" and "his speech almost never made sense." He had to be shown the way to the bedroom and bathroom, and incontinence was frequent.

Examination on admission disclosed a cooperative, alert man with blood pressure of 190/110 mm Hg. He was oriented as to place and person, but not to time. He laughed when he encountered speech difficulty. Speech was fluent and circumlocutory, and filled with many literal and verbal paraphasias. Words were well-articulated, but there was halting between phrases. When asked "What is wrong with you?" he replied after long hesitation and with marked gesturing and great effort "...I cannot find my words." At times he would state "I know what I mean." Comprehension was intact. He correctly pointed to the "source of illumination in the room" and to "the place where people enter the room." When asked if he were in the Massachusetts General Hospital, he answered "no", and answered "yes" when asked if he was in the Boston City Hospital. His replies to questions demanding a "yes" or "no" answer clearly indicated that his comprehension was intact. Repetition was severely impaired. He could occasionally repeat single monosyllabic words such as "bed", but had extreme difficulty with multisyllabic words such as "constitution" and "hippopotamus". There was a tendency to approximate the sound of the first syllable, often resulting in a neologism. "President" became "peh-tn". On repeated

attempts the result was less and less recognizable. He could not repeat "no if's, and's, or but's". Series speech was relatively preserved. Days of the week were slowly but accurately recited. Serial digit repetition was better preserved than verbal repetition. Naming was poor. He named a pencil but failed to name a watch, tie, shirt, and other objects. Color naming was poor.

The patient was apraxic. Although he protruded his tongue to command, he had great difficulty executing commands such as "show me how to suck from a straw", "show me how to brush your teeth", and "comb your hair". Performance improved somewhat with imitation, and was often better when the real object was used. Thus, in pretending how to use a hammer, he tended to use the body part as the object and failed to improve with imitation or with use of the real object. Although the patient could recognize and read individual letters, he could not read aloud. He was unable even to write his name. Finger recognition was extremely poor, right-left orientation was impaired, two-point discrimination was poor bilaterally, and spatial disorientation was profound. Calculation could not be performed. He could copy a square, but not a cube. He drew a clock but inserted letters and symbols rather than numbers.

The fundus was normal on the right, but a dense cataract obscured the left. There was no useful vision on the left, and the acuity was 20/200 on the right. Other cranial nerves were normal. There was mild weakness of the intrinsic muscles of the right hand, but strength was otherwise intact. Deep tendon reflexes were increased on the right side, and the right plantar response was abnormal. His gait was slow and uncertain. Sensory examination was intact to all primary modalities.

Lumbar puncture yielded clear CSF under normal pressure. The CSF contained two red blood cells per cubic millimeter and the protein content was 151 mg/100 ml. Blood glucose levels ranged to 400 mg/100 ml and were controlled with insulin injection. An electrocardiogram revealed changes consistent with a posterior wall myocardial infarction. Three days later, he had a cardiopulmonary arrest but was promptly resuscitated. There was no change in his neurological status. One month after admission, he suffered extension of the myocardial infarction and died.

At postmortem examination, the heart weighed 400 gm. The coronary

arteries were moderately atherosclerotic, and there was complete obstruction of the left anterior descending coronary artery 1.5 cm from its origin. There was a large myocardial infarction in the territory of the occluded vessel.

The cerebral arteries were moderately atherosclerotic. There was an old occlusion of the parietal branch of the left middle cerebral artery due to thrombus formation on an atherosclerotic plaque. Softening was present over the left supramarginal and angular gyri areas in the territory of the occluded vessel. The brain, which weighed 1,270 gm, was fixed in 10% formaldehyde solution, and cut at 0.8-cm intervals (Figure 3). In

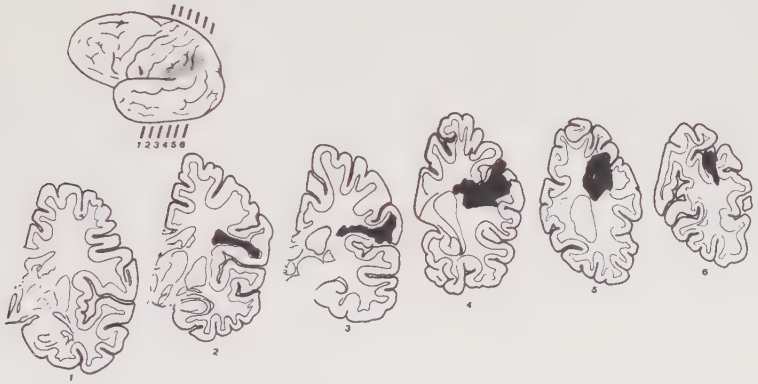

Fig. 3. Indication of extent and location of infarct (case 3).

the left parietal lobe, there was an irregular cavitated infarct that measured $3.4 \times 2.2 \times 4$ cm. This lesion involved the white matter beneath the supramarginal and angular gyri. The cortex in these areas showed several small infarcts. The softening extended in a wedge-shaped pattern to the body of the left lateral ventricle, interrupting the arcuate fasciculus. The infarct appeared to be more recent in its more posterior parts. Small lacunae were identified, one in the right corona radiata and another in the left internal capsule. Both the Broca and Wernicke areas were entirely intact.

II. COMMENT

The language performances in these cases were remarkably similar.

All three patients were definitely fluent, but the verbal output was contaminated by paraphasias which were predominantly literal (i.e., consisting of phonemic substitution). All performed poorly when attempting to repeat despite excellent comprehension. Thus, the primary characteristics of conduction aphasia were present. While the patients were not evaluated during the earliest phases of the disorder, the reports available suggest that comprehension of spoken language was always at a high level. Normal auditory language comprehension was always demonstrated by both clinical and psychological testing. The first two patients improved in spontaneous speech and in repetition, but neither of these functions returned to even near-normal. The theory that conduction aphasia necessarily represents a stage in the resolution of Wernicke aphasia (with comprehension improving first) would not seem to be supported by these findings. Similarly, the fluency of their conversational speech and the quality of the verbal content (many filler words, few substantive words) distinguishes the expressive disturbance of these cases from that seen in Broca aphasia. Their language disturbance represents a distinct clinical syndrome, readily separable from other forms of aphasia, that deserves identification by a separate name (Benson and Geschwind, 1971).

While the similarity of the language performance of these patients was striking, there were differences in other aspects. Most obvious was the presence of well-defined ideomotor apraxia in the first and third patients and the total absence of apraxia in the second. This difference was not simply a matter of degree. The apractic disturbance was so dramatic in case 1 that he served as the subject of a movie used to illustrate a discussion of apraxia presented to the annual meeting of the Academy of Aphasia (Kaplan, 1962). Case 2, on the other hand, was carefully and critically evaluated by many examiners who consistently failed to demonstrate any disturbance of praxis.

The findings at postmortem examination also showed significant differences. One can discount the large right hemisphere lesion in case 1 which accounts for the left hemiplegia that appeared long after the onset of aphasia. There were a number of smaller lesions in the left hemisphere. Of these, the lesion that most likely related to the language disturbance and apraxia involved the white matter of the supramarginal gyrus, including the arcuate fasciculus, and the overlying cortex. A

similar lesion was present in case 3. In both of these cases the infarct lay entirely above the sylvian fissure, totally sparing the Wernicke area. In contrast, the lesion in case 2 involved the entire first temporal gyrus of the left hemisphere, obliterating all auditory association cortex. Patients 1 and 3 had no lesion of significance in the region of the auditory association cortex while the second patient had little involvement of the supramarginal gyrus or the arcuate pathway deep to this gyrus. There would appear to be two distinctly different neuroanatomical localizations underlying the essentially identical language disorders.

The world literature includes only a few reports of 'conduction aphasia' that have postmortem correlation (see Table I). There is some difficulty in the interpretation because the reporting of clinical and postmortem findings is not standardized. Most of the reports describe lesions that involve both the posterior part of the superior temporal lobe and the inferior part of the supramarginal gyrus, the major abnormality involving the posterior portion of the perisylvian region both above and below the fissure. Several of the reported cases, however, manifest pathological findings that are similar to those in our first and third cases (Pershing, 1900) while in several others (Liepmann and Pappenheim, 1914; Stengel, 1933) the findings more closely resemble those in our second case. Thus, it would appear that pathological involvement of either of two, neighboring but different anatomical sites may produce conduction aphasia.

Our patients 1 and 3, who had lesions involving white matter and cortex of the parietal operculum, represent examples that support the interpretation of a disconnection between Wernicke and Broca areas. According to this hypothesis, the intactness of the Wernicke area accounts for normal auditory-verbal comprehension, while the intactness of the Broca area accounts for well-articulated fluent speech. The lesion of the parietal operculum, however, blocks transmission from the Wernicke to Broca area so that repetition is poor. The net effect is a fluent aphasia with good comprehension but poor repetition. A similar interpretation is given by Konorski *et al.* (1961) who refer to this syndrome as audioverbal aphasia.

Our second case, however, appears to correspond to a mechanism advanced by Kleist (1934). He argued that, in some patients, destruction of the Wernicke area does not lead to the severe disorder of auditory comprehension that is a characteristic of lesions in this site, because the

right hemisphere takes over the function of auditory comprehension. He further assumes that in these patients the Broca area (on the left side) is still required for the production of speech. For repetition the heard words must be transferred from the right-sided homologue of the Wernicke area across the corpus callosum to the corresponding region on the left and then forward to the Broca area via the arcuate fasciculus. The left temporal lesion, however, destroys one end of the callosal pathway and repetition is therefore impaired. Although Kleist argues that this is the only mechanism for conduction aphasia, his own diagram included the left arcuate fasciculus as the final pathway to the Broca area. This would imply that a lesion of the arcuate fasciculus should be capable of producing the same syndrome. Indeed a review of Kleist's (1962) own material suggests that he too observed cases of this type. His patient 2 showed gross destruction of the left temporal speech area, but his fourth patient had damage predominantly in the parietal lobe.

It seems likely that in some patients the right hemisphere can rapidly assume the functions of comprehension after destruction of the Wernicke area. Alternatively one might assume that there are some patients in whom the right side is normally dominant for comprehension, while the left is dominant for expression. However, this latter conjecture seems less likely in view of the rarity of comprehension loss after purely right temporal lesions. Why the right side can function in this manner in such patients as our case 2 remains a mystery. It is of note that this patient had one left-handed child. It is also of interest that Kleist's patient, although right-handed in several activities, was clearly left-handed in others, e.g., fencing. Luria (1970) has emphasized the better outlook for recovery from aphasia after left-sided lesions in those who are left-handed or have left-handed relatives.

A recent study by Kinsbourne (1971) supports the presence of right hemisphere auditory comprehension in some cases of conduction aphasia. He reports that after a right carotid amobarbital (Amytal) injection a patient with the clinical findings of conduction aphasia suffered a distinct but temporary loss of comprehension. A similar injection on the left side did not disturb comprehension, supporting Kinsbourne's conclusion that the right hemisphere was performing the task of auditory comprehension in his patient. Several years ago we performed carotid amobarbital injection on the left side of a patient with conduction aphasia and

produced a clearcut disturbance of comprehension. It would appear that he was using the Wernicke area on the left for language comprehension. In this patient, a brain scan performed soon after onset showed a well-defined uptake deep in the left posterior suprasylvian area, suggesting a pathological picture similar to that in cases 1 and 3 of this report. These two instances of amobarbital-produced comprehension deficit after injection of different sides add further support to the present postulation of two distinct pathological bases for the same aphasic syndrome.

The presence of apraxia in our patients 1 and 3, contrasted with the absence of apraxia in patient 2, deserves comment. One might conclude that the same disconnection that produced the repetition disturbance also produced the apraxia in these patients. They understood the verbal commands but were unable to pass the messages forward to the motor area for enactment. Why, then, did our second patient not have apraxia?

One possible explanation is that he could carry out the commanded motor activities by using pathways in the right hemisphere. From the right hemisphere analogue of the Wernicke area there are connections forward to the right motor association cortex. Here callosal pathways exist that connect to the left motor association cortex. Thus, one can postulate an alternative pathway for the conduction of verbally commanded motor activities. If this explanation is true, however, why do these pathways fail to function for the repetition of speech? At present we can offer no simple answer to this question.

A second possible explanation of the discrepancy in apraxic symptoms can also be mentioned. As well as longer fibers, the arcuate fasciculus contains short fibers that synapse en route. It is not clear at this time whether the long fibers carry verbal commands directly to the premotor region or whether a synapse in the parietal operculum is involved (Geschwind, 1965). If such a synapse were necessary then, even if short fibres from the Wernicke area were destroyed, the cells of the parietal operculum on which they synapse would be intact. These cells would be available to receive verbal commands across the corpus callosum for transmission forward to the premotor region if comprehension were accomplished in the right hemisphere. This alternative explanation, however, still leaves unanswered why this alternative pathway is not also available for the repetition of speech. The data available from these cases fail to resolve this question.

The presence of apraxia in facial and limb movements may represent an important clinical feature for distinguishing the two forms of conduction aphasia. We propose that (1) the presence of apraxia in a case of conduction aphasia supports the presence of a parietal opercular lesion; and (2) cases of conduction aphasia with lesions only in the Wernicke area will not manifest apraxia.

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CHAPTER XXVI

APRAXIA AND AGRAPHIA IN A LEFT-HANDER

PREFACE

As I pointed out earlier disorders of writing may occur as the only or predominant linguistic deficit in patients with confusional states, an apparently paradoxical and totally unexplained finding. Oddly enough isolated writing disorder also tends to occur, as Hécaen has pointed out, in left-handers, or may remain as the only linguistic disorder after recovery from other disturbances, as in the case of Dejerine and André-Thomas. The case presented in this paper permitted some speculations as to the mechanisms of this curious phenomenon.

The study of left-handers is important precisely because of their tendency to recover better from aphasia than right-handers, a fact documented extremely well by Luria in the book reviewed in the previous paper.

APRAXIA AND AGRAPHIA IN A LEFT-HANDER*

1973

ABSTRACT. Although agraphia of the right hand, in the absence of other significant language disorders, has been described in left-handed patients, there has been no full explanation of these phenomena. This paper presents a case of a left-handed patient who developed a left hemiplegia without aphasia, and who not only lost the ability to write with the right hand (which he had always used for this task) but also showed apraxia on the right. Analysis of this case suggests the presence of language dominance in the left hemisphere and dominance for handedness in the right hemisphere. Since handedness and language were in different hemispheres the corpus callosum was probably traversed in the normal act of writing. The thesis is advanced that a lesion in the hemisphere dominant for handedness destroyed the engrams for complex motor activities and was responsible for this patient's agraphia and apraxia.

Loss of the ability to write with the right hand in the absence of other significant language disorders occurring in patients who were left-handed but had always written with the right hand has been described in several case reports (Dejerine and André-Thomas, 1912; Nielsen, 1946). Others have also commented upon the relatively high frequency of cases of pure agraphia among left-handers (Dubois *et al.*, 1969). There has, however, been no full explanation for these phenomena. We present a case of a left-handed patient who developed a left hemiplegia without aphasia who not only lost the ability to write with the right hand (which he had always used for this task) but also showed clear-cut apraxia of the right side. We think that the analysis of this case may not only help to elucidate other cases in the literature but also help us to understand more about cerebral organization in left-handers and about cerebral dominance.

I. CASE REPORT

L. T., a 62-year-old man with a history of hypertension, suddenly had left-sided hemiparesis six days before first being seen by us. The patient noticed some slurred speech, but he could speak with correct grammar and choice of words. Before this episode he had always written with his right hand but performed most other activities with his left hand: cutting,

throwing, using a toothbrush, hammer, razor, or comb; he usually used his left eye to shoot and his left foot to kick. The patient recalled being left-handed as a boy and having his writing switched to the right hand while he was in school. One brother was left-handed, but four other siblings and both parents were right-handed.

The history was also pertinent in that the patient said that one year before he had had transient weakness of his right arm which lasted about fourteen hours. After this, he had no known difficulty with writing (Figure 1A). Two years before he had an acute myocardial infarction.

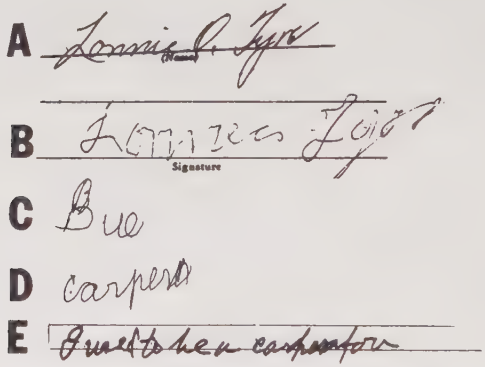

Fig. 1. (A) Patient's signature (written with right hand) before onset of left hemiparesis. (B) Patient's signature with right hand after onset of left hemiparesis. (C) and (D) During hospitalization patient was asked to write, in a full sentence, his occupation. He tried to write 'builder' but was unable and then tried to write the word 'carpenter'. (E) Six months after discharge he was again asked to write his occupation.

On admission his temperature was 100.4 °F, blood pressure 118/78, and pulse 120. His haematocrit was 51 percent and his BUN was 32 mg per cent. He was noted to be 'confused' and many mental inconsistencies were noted. With adequate hydration, he became afebrile, his haematocrit returned to 41 mg per cent and BUN to 12 mg per cent. Thirteen days after admission he was alert and co-operative.

The patient had a right superior quadrantanopsia and except for some left lower facial weakness, the cranial nerves were normal. He had a left hemiplegia involving both the upper and lower extremities; his deep tendon reflexes were slightly brisker on the left than on the right, and he

had a left extensor plantar response. Sensations of touch, pin, vibration, hot and cold all felt different on the left side, though the stimulus was recognized. Stereognosis, two-point discrimination and tactile localization were grossly impaired on the left. The patient knew the date but was not certain in what city he was although he knew he was at the Veterans Hospital. The last President he could remember was Mr. Johnson and his memory before that was good. He knew that there was a war in Asia but did not know in what country. He was unable to recall any of three objects after five minutes, but on retest he was able to recall two out of the three objects. His remote memory was excellent. The patient stated that an apple and a banana were both food, and a car and a train both had wheels. When asked to explain "a stitch in time saves nine", he stated, "one stitch now will save nine later". He was able to do simple calculations and to distinguish right and left both on himself and on the examiner, and was able correctly to follow commands such as "put your left hand on your right ear". He knew his fingers and could copy a cube.

Spontaneous speech was normal with no evidence of paraphasia or any other language disturbance. Comprehension was intact as was ability to name even low frequency objects such as a watch crystal, watch band, and buckle. He could repeat quite well, even sentences such as "no ifs, ands, or buts". He was able to read out loud quite well and was able to comprehend written material.

In striking contrast to the normal language performances, difficulties in writing were prominent. Spontaneous writing with the right hand was extremely poor (Figures 1C and D), and he even had difficulty writing his own name (Figure 1B). His writing to dictation was also poor, but his writing improved with copying. Because of the left hemiplegia, writing with the left hand could not be tested. The patient's ability to spell words aloud was normal, and when words were spelt aloud to him, he identified them correctly. His ability to type was difficult to interpret because he showed neglect of the left half of the keyboard.

When asked to blow out a match, to stick out his tongue, and to show how to suck on a straw to command, he was able to make these movements properly and showed no evidence of a buccofacial apraxia. However he had severe apraxia in the right arm. When asked how he would brush his teeth, he brought up his hand to his mouth as if he understood the command, then he kept his fist closed and rubbed it against his

closed lips. When asked to show how he would comb his hair, he brought up his hand to his hair, and then used a fist to straighten his hair. Similarly when asked to use a hammer, throw a ball, flip a coin, he would in each case make a motion but in no case did he correctly carry out these commands. When these actions were demonstrated by the examiner, the patient was unable to improve his performance to command; although, with the actual object there was an improvement.

Although he was aware of his severe hemiparesis, it did not appear to concern him. On testing with simultaneous auditory, somaesthetic or visual stimuli the patient showed extinction on the left side.

Laboratory studies included a brain scan which showed an abnormal uptake in the right hemisphere, and an EEG which showed slowing over the right hemisphere. The CSF had normal pressure, protein, cells, sugar, and a negative test for syphilis. Skull X-rays were normal.

The patient was in hospital for fifty-one days whilst his agraphia and apraxia gradually improved. Six months after discharge, although remarkably improved, the patient showed a mild but definite apraxia and agraphia in the right limbs and the left hemiplegia persisted.

II. DISCUSSION

Liepmann and Maas (1907) were the first to show that unilateral apraxia and agraphia in the absence of aphasia, similar to that seen in our patient, can result from a disconnexion of the two cerebral hemispheres, a result confirmed by other German authors in the first years of this century. In more recent years Geschwind and Kaplan (1962) described a similar patient, and essentially the same results were found in later cases by Gazzaniga *et al.* (1962). The lesions in these disconnexion syndromes involved the callosal fibres. In most of these reported cases there was an agraphia and apraxia of the left hand but not the right hand, an inability to name objects placed in the left hand but a retained ability to identify the object non-verbally. In addition, a unilateral grasp reflex and a unilateral paresis of the lower extremity were sometimes associated with the callosal disconnexion syndrome, reflecting involvement of other portions of the anterior cerebral artery distribution.

In the reported callosal cases the patients were right-handed and the agraphia appeared in the left hand, i.e. the one ipsilateral to the hemi-

sphere dominant for speech. It is therefore conceivable that a callosal lesion could cause an agraphia of the right hand in someone who was right-brain dominant. Indeed it seems to be clear that such cases exist. Thus the case of Nielsen (1946) was a left-handed man who had always written with the right hand. He suffered a stroke in which he developed weakness of the right leg, but not the right arm, but he lost the ability to write with the right hand but was found to be capable of writing with the left hand. In view of the hemiplegia which involved the leg primarily it seems likely that this stroke was in the anterior cerebral distribution, and that the anterior four-fifths of the callosum were also infarcted.

The famous case of Pitres (1884) is often cited as a classical example of pure agraphia, but Pitres' patient was agraphic with the right hand only and could write with the left hand. No description is given of his handedness and the neurological examination is not given in detail, but he had increased reflexes in the right leg. It is again possible that this case is an example of the same mechanism as Nielsen's case.

It seems unlikely, however, that this mechanism could account for our patient's state. The sudden onset of a hemiparesis which severely involved the upper and lower limbs and the presence of neglect suggest that the lesion was probably in the distribution of the middle cerebral artery rather than the anterior cerebral artery. Furthermore, if there were primarily a callosal lesion, one would expect incorrect identification of objects in the right hand, not the left, as was demonstrated in our patient. The astereognosis and other defects of cortical sensory function probably resulted from a right parietal defect. Finally, brain scan confirmed a lesion in the distribution of the middle and not the anterior cerebral artery.

A further powerful argument against the idea that in this patient one is dealing with callosal disconnexion leading to agraphia in the hand controlled by the hemisphere which is not dominant for speech is evidence which suggests that in fact the left hemisphere was the one which was dominant for speech. The brain scan strongly supports a large lesion in the centre of the right middle cerebral artery distribution. If the right hemisphere had been dominant for speech it is difficult to see how an aphasia could have been absent. We must thus explain the curious phenomenon of an agraphia in the hand controlled by an intact hemisphere which is dominant for speech.

The same reasoning permits us to exclude the possibility that this case is identical to the case recently reported by Poeck and Kerschensteiner (1971) in which a right hemisphere lesion in a left-handed patient led to a left hemiplegia and apraxia in the right hand. Poeck's patient was, however, aphasic so that we have evidence that the right hemisphere was involved in speech, which was not the case in our patient. Poeck's case is, of course, similar to the common finding, in patients with Broca's aphasia and right hemiplegia, of a left-sided apraxia. In these cases the left-sided apraxia is also probably the result of destruction of callosal fibres at or near their site of origin in the cortex, thus disconnecting the right hemisphere from the left.

Another possible explanation would be that the patient had another lesion in the left hemisphere to account for the right-sided agraphia. The patient had right upper quadrantanopsia, which was probably related to the transient right hemiparesis that occurred one year before. The lesion responsible for this quadrantanopsia was probably a small lesion in Meyer's loop or an infarct of the lower lip of the calcarine fissure. The production of pure agraphia by focal lesions is at best extremely rare and either of the above localizations would appear unlikely. Furthermore, the patient did not become agraphic until the occurrence of the left hemiplegia. Although some cases of pure agraphia have been attributed to lesions in the so-called area of Exner, most of the cases in the literature have had tumours, raising other possibilities of interpretation, to which we will allude later. There is no clinical EEG or brain scan evidence to support a frontal lesion and thus the possibility of a focal left frontal vascular lesion seems exceedingly unlikely.

Another possibility which might be raised was that the patient had a second lesion not frontally, but in the left parietal region, and that his agraphia was simply part of a Gerstmann syndrome. If so, one might have expected at least some of the other components of the syndrome, i.e. finger agnosia, acalculia or right-left disorientation but all were absent as was alexia. The absence of constructional apraxia would also be very unusual. Furthermore, our patient had a significant right-sided ideomotor apraxia which is not a usual accompaniment of a Gerstmann syndrome. In summary, a second left hemisphere lesion producing a partial Gerstmann syndrome seems very unlikely.

One further mechanism of agraphia without other language disorders

deserves mention. One of us (N. G.) has, for many years, noted the presence of agraphia without other language disorders in patients with acute confusional states, such as delirium tremens, or metabolic encephalopathy. Chédru and Geschwind (1972) have documented this finding in a large series, and have pointed out that most cases of so-called pure agraphia in the literature have had large tumours or have been described as demented, raising the possibility that the agraphia was not the result of focal disease but rather of raised intracranial pressure or diffuse disorder. This mechanism seems unlikely in our patient since he was alert, co-operative and attentive and was perfectly capable of maintaining a coherent stream of thought. Although he did show some deficits on mental examination, his total mental status was clearly different from that of the patients in whom agraphia has been observed during confusional states. This mechanism therefore seems unlikely.

Our task in this case is therefore to account for the presence of agraphia in the hand opposite a hemisphere which still serves speech, following a lesion in the hemisphere ipsilateral to that hand. It is of major importance that in the other similar case in the literature there was a left hemiplegia in a left-handed woman as the result of a lesion in the right hemisphere.

The case of Dejerine and André-Thomas (1912) is similar to ours, although there are differences which in themselves we feel may be most illuminating. Their patient was a 49-year-old woman, left-handed for nearly all activities, who had, however, written with the right hand. She had severe hemiplegia, hemisensory loss and hemianopia, all on the left, at first with a clear-cut aphasia and incomprehension of written language whilst understanding spoken language well. She remained in the hospital until her death five years later. At the last examination her general intelligence was clearly excellent. Spontaneous speech was now nearly normal but repetition, particularly of phrases containing prepositions, was poor. Reading comprehension was now near normal. She still showed, however, marked agraphia, both spontaneous and to dictation, but copied perfectly, even copying print into script. Thus, although this patient began with a left hemiplegia and aphasia, by the end of her illness she had a left hemiplegia with agraphia in the intact right hand (the left could of course not be used) as the striking disorder of higher functions. There is no mention of praxis testing.

At post-mortem the brain studied in serial sections showed no lesions at all in the left hemisphere, but there was a massive infarction involving all lobes in the right hemisphere and the right side of the corpus callosum. It thus is clear that the return of language functions must have been subserved by the left hemisphere. The patient thus showed at the end a picture similar to that of our case, i.e. agraphia in the hand opposite an intact left hemisphere which was capable of other language functions.

Let us now turn to the possible interpretation of these cases. We must first consider something which is very often neglected, exactly what is

meant by handedness. Clearly we can exclude greater strength of one arm as the cause. Similarly, although dexterity is normally greater in the dominant hand, one must keep in mind the possibility that dexterity is the result of preferred use, rather than vice versa. That this may be the case is seen when one considers the occasional patient with infantile hemiparesis who continues to prefer to use the hemiparetic arm, despite its deficiencies in strength and elementary dexterity (Hécaen and Ajuriaguerra, 1964).

A possible hypothesis is the one offered by Liepmann (1908), i.e. that manual dominance reflects the ability of one hemisphere to learn motor tasks more readily than the other. In support of this view Liepmann advanced powerful arguments. Thus, in patients with callosal lesions, either in mid-callosum or with lesions in cortex destroying the origin of the callosal fibres in the dominant hemisphere, one finds not only failure to carry out verbal commands with the minor hand, but very frequently failure to imitate the movements made by the examiner and in some instances failure even in handling objects with that hand. This occurs in the presence of a fully intact minor hemisphere in a fully alert and co-operative patient. Liepmann argued that these facts show that the minor hand operates under the control of the dominant hemisphere, exerted across the corpus callosum. Since its skill is borrowed from the opposite hemisphere it becomes even less skilful than it normally would be after such lesions. A recent paper by Wyke (1971) lends support to the Liepmann view. She showed that patients with left hemisphere lesions do more poorly on a test of learning a bi-manual co-ordination than those with right hemisphere lesions.

If the Liepmann analysis of handedness is correct then the presence in some people of language dominance in one hemisphere and dominance for handedness in another seems reasonable. In our patient left hemisphere dominance for language appears almost certainly to be present. In the patient of Dejerine and André-Thomas it seems likely that although the patient was initially aphasic from a right hemisphere lesion there was enough bilateral representation of language so that the left hemisphere eventually could function normally in this respect.

The evidence in right-handers, like those cited earlier, is that writing with the left hand is under the control of the left hemisphere by way of the corpus callosum. Writing is, however, presumably different from the

other modalities of language in that it needs both linguistic capacity as well as the learning of complex manual motor control. Whilst these are both housed in the left hemisphere in right-handers, these abilities may be in different hemispheres in the left-hander. If a lesion destroys the engrams for such complex motor activities, then the patient will be agraphic and apraxic, as in our case. Clearly it would have been of great interest to have had some definite information about praxis in Dejerine and André-Thomas's case but this is not recorded. The fact that our patient could draw and copy does not necessarily present a difficulty, since impairment in this type of task may be absent in patients with apraxia and may be present in patients who are not apraxic.

The thesis presented here, if correct, would carry certain implications which would be subject to confirmation. Thus it would seem that the process of writing in some left-handers may be very complex. If the patient is right-brained for complex motor sequence learning (as indeed should be the case in the normal left-hander) and he is right-brained for speech then left-handed writing would require no callosal participation. A callosal lesion in such a patient should produce right-sided agraphia and apraxia. If he is right-brained for speech and handedness but writes with the right hand then the left hemisphere is presumably controlled across the corpus callosum. He too should be agraphic and apraxic with the right hand after a callosal lesion but should be able to write with the left hand.

If the patient is left-brained for speech, but right-brained for handedness and writes with the right hand, then it seems reasonable to speculate that in the normal act of writing he must traverse the callosum twice, i.e. he must relay the linguistic information from left to right to arouse the appropriate motor sequence and then must relay the motor sequence back to the left motor regions to control the right hand. If he writes with the left hand he need traverse the callosum only once. One might speculate as to whether the left-handers taught to write with the right hand who later reverted to left-handed writing (Gloning *et al.*, 1969) were largely within the group who required double callosal transfer for right-handed writing.

The effects of a callosal lesion in the left-speech dominant, right-motor dominant cases might well be quite complex. The patient should be apraxic and agraphic in the right hand (because of disconnexion from the motor-dominant hemisphere). He should be agraphic in the left hand

because of disconnexion from the speech-dominant hemisphere. He should be apraxic in the left hand but only to verbal command, preserving imitation and object handling. All these analyses would of course be modified if after the lesion the patient could shift to the other hemisphere for either speech or motor control.

It is worth specifying here the possible complexity of the situation in the case of a pathological left-hander. This analysis suggests that in some pathological left-handers motor dominance has shifted to the right hemisphere, but it is conceivable that in others motor dominance may have remained on the left side, but that the use of the left hand reflects the difficulties in the use of a weak right hand. It is conceivable that in either case speech dominance may or may not shift. There are thus many possibilities for the effects of lesions in such brains.

One further point should be made about the apraxia in our patient. Hécaen and Sauguet (1971) reviewed a series of 73 left-handers. There was a remarkably low incidence of ideomotor apraxia in these patients with left-sided lesions and none with right-sided lesions. This may be the result of the time of examination of the patients. Obviously apraxias do occur in left-handers as the recent report of Poeck and our own case show. It should be noted, however, that both in our case and Poeck's the apraxia (and in our case the agraphia) improved with time. It may be that left-handers have a better recovery than right-handers from apraxias just as they do with aphasias. If this is the case, our analysis would suggest that the left-hander may commonly show the same capacity to shift hand dominance after an adult brain injury as he shows for speech dominance.

A comment is appropriate concerning the discussion of Leonhard (1952) of agraphias after right-sided lesions, since his interpretation which bears some resemblance to ours, differs in important ways. He argued that since at the time of his writing, among Europeans the right hand was almost always used for writing, even by left-handers, the "writing centre" was in the left hemisphere. He went on to argue that the cases who became agraphic in the right hand with right hemisphere lesions were actually people who were right-dominant (presumably for language). The effect of the right hemisphere lesion was to cut off the language-dominant hemisphere from the writing centre. We would differ from him in his assumption that the left hemisphere necessarily develops, on the basis of training, special capacities for writing. His interpretation furthermore

would not seem to us to account for the case of Dejerine or for our own case in which the patient was agraphic despite the evidence that the left hemisphere was functioning in language.

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